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PARASITIC FUNGI INTERNAL OF SEED CORN[1]

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The importance of root, stalk, and ear rot fungi in decreasing yields of corn has received considerable attention in recent years on the part of investigators. Results of investigations so far reported indicate more or less agreement in the various disease symptoms manifested. However, some difference of opinion exists concerning the importance of the causal organisms. The specific determination of the fungi has not been fully emphasized nor the method by which they are carried in the seed.

The following report presents in part the results of our investigations in determining the species of fungi associated with corn rots. Our studies were initiated to ascertain the losses to the corn crop and prevalence of infection in Delaware and the importance of the seed in carrying infection. While our observations and studies have been confined principally to field corn in this State, we feel that careful investigations will reveal the presence and importance of the same pathogenes in other States but varying somewhat in prevalence.

A review of the literature covers the observations and investigations conducted in this country so far as we have been able to secure them. In this review only special mention is made of results bearing directly on seed infection, the fungi reported, and the method by which the fungi are carried in the seed. Because of the extensive observations reported on the root, seedling, and stalk infection it was deemed best to discuss this phase of the problem in a subsequent paper to be prepared by the writers.

"Moldy corn" no doubt was one of the first symptoms observed in connection with corn rot diseases. The presence of mold in corn was early associated with forage poisoning and pellagra in this country, as well as in Europe. Starting with these early observations were studies which were later extended to include and establish the importance of the fungi which are now associated with the corn rots.

Sheldon (*29*)[2] described *Fusarium moniliforme* Sheldon, associated with moldy corn and thought to be the cause of forage poisoning. He observed—

> the kernels where the pink mould was present were considerably broken at the ends and crumbly so that when shaken or struck on the table, the inside would fall out in

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[2] Reference is made by number (italic) to "Literature cited," p. 523-524.

the form of a coarse powder; it attacks the end of the kernel first, gradually working its way toward the cob.

Valleau (*36*) in his studies of corn rots in Kentucky as well as those from other States, considers that *Fusarium moniliforme* will probably prove to be the most common cause of root and stalk rots of corn. He finds that—

a microscropical examination of the pink crowns or pink stripes on kernels reveals the presence of hyphae and occasionally spores in them, between the seed coats. The development of reddish or black discoloration in the seed coats of corn during and after germination, is an indication of infection with *F. moniliforme.*

No mention is made of the importance or determination of other fungi in connection with his studies. In a later article (*37*) on the control of corn root rot, it is stated that—

thus far no disease free seed has been obtained by the method of selection. The results obtained indicate that infection takes place before the early dough stage. It probably occurs through the silks and is a result of infection of the exposed silk mass with *F. moniliforme.* Attempts to control corn rot by seed treatment have given negative results.

Barrett (*5*) reported on the dry rot in corn as caused by several species of fungi. One of the most destructive being *Diplodia maydis* Sacc. (*D. zeae* (Schw.) Lev.). A second form of dryrot which is not uncommon is due to a species of Fusarium, and a third form is due to a sterile fungus. Later in conjunction with Burrill (*6*) studies were reported on ear rots of corn. Four types of ear rots were recognized as caused by *D. maydis* and three different fusariums, designated 1, 2, and 3.

The most extensive work was on *Diplodia zeae.* They observed—

the slender threads penetrate the young tissue of the grains, cob and husks, progressing from cell to cell and extracting from their contents whatever is of value for food. Diseased ears left in the field under natural conditions eventually develop numerous pycnidia in the grains, giving them a black appearance.

The best results of inoculations were obtained when the corn was still in the thick milk stage.

80% of silk inoculated ears produced the disease, 71.7% of those inoculated at the base and 48% so treated in the shanks were successful, while but 22% of the sprayed ears showed any signs of infection. All later inoculations altho fairly successful produced smaller percents of the disease than others.

There was no direct evidence obtained that the infected ears on inoculated stalks were a result of the inoculations made.

Fusarium 1—

produces a rather dense, felty mass of white mycelium which extends between the kernels to the cob, causing it to become more or less diseased.

The mycelium is established all through the diseased grains, corroding the starch and destroying the germs.

The diseased portion of ears infected with Fusarium 2 have a deep pink to red color due to the pigment produced in the hyphae of the fungus.

The felty mass of mycelium permeates the inner husks and silk and holds them firmly to the ear.

Fusarium 3 forms a rot less complete in its destructiveness of the ear than that of the other forms described. Many of the infected ears have only a few scattered disease grains.

Under some conditions, however, most of the kernels may become diseased and the cob more or less infected . . . . The mycelium is white, very sparse, and is

found principally in the end of the kernels where it feeds upon the starch and produces large numbers of spores, mostly microconidia.

Corn inoculated in the thick milk stage shows the largest percentage of infected ears in the field.

It is worthy of remark that in no case has natural infection by these parasites been discovered upon any other part of the immature corn plant save the ears and their belongings. Upon the latter infection always begins externally from air distributed spores.

The parasitism of *Diplodia zeae* has been more thoroughly established than any of the other fungi associated with corn rots. Heald, Wilcox, and Pool (*16*) state that this fungus produces in the ear a condition which may be called "dry rot."

In the early stages or in cases of slight attack, no external evidence of the presence of the fungus can be detected. It is probable that the hyphae enter the kernel at its base, since the fungus is first detected in this region. From this point the hyphae grows throughout the kernel and are found in both the endosperm and embryo.

They consider the prevailing type of infection is through the silk near the time of pollination. This disease was observed by Stevens and Hall (*33*) in North Carolina where it is known as "mold," "mildew," "rot," and "souring."

The disease affects the ear, manifesting itself as a whitish growth of mold over the surface of the grains, sometimes affecting the whole ear and at other times portions only of it. The amount of fungus visible upon the superficial parts of the grain is not large, but upon breaking open the ear, it is found that the spaces between the bases of the kernels are often densely packed with masses of pure white mycelium. Externally, no signs of fruiting bodies of any kind are apparent, but close examination at the point of attachment of the grains to the cob, in many instances, reveals the presence of exceedingly minute black specs, which under the microscope prove to be the fruiting organs (pycnidia) of the fungus.

They determined the fungus as *Diplodia macrospora* Earle, but *Diplodia zeae* Lev. also was found in several instances. Smith and Hedges (*31*) concluded on the basis of pot experiments that the manner of infection indicated is the common one, that is—

from the soil into the roots, from these to the interior of the stems and thence upward to the cobs, and finally to the kernels.

Evans (*11*) reports maize cob mold (*Diplodia maydis Sacc.*) as prevalent in Natal. It is also frequently referred to as mildew.

Many of the farmers showed me samples of mildewed maize cobs which they stated caused them severe loss in their crops, and also produced severe paralysis and frequently death amongst stock that were fed on these cobs, especially if the cobs were damp and not properly dried out.

Van Der Bijl (*38*) conducted extensive studies on the "Dry-Rot" of maize in South Africa and considers that normally infection takes place through the silks. A Fusarium is also mentioned as being found several times with moldy corn. Garman (*12*) states that—

Diplodia zeae is not dependent on the openings made by the worm, generally invading ears by way of the shank.

Fusarium-like fungi for the greater part were associated with these corn diseases by the early investigators. It was not until recently that several recognized species of Fusarium were definitely established with the root, stalk, and ear rots of corn.

Garman (*12*) along with his studies of pellagra mentions Fusarium and *Tricothecium roseum* associated with moldy corn. He states that—

seed corn should be inspected for molds with special care, a pink mold (a *Fusarium*) is very common in our fields and causes many grains apparently sound to assume a

pink color. What appears to be the same fungus is on corn that is germinating badly in the field.

Arzberger (4) investigated the cob rot of corn caused by *Coniosporium gecevi* Bubák. It is reported that—

Coniosporium has an economic significance in that it destroys the cob tissue as a saprophyte; its effect on the kernels is rather limited when compared with the injury of *Diplodia*, *Fusarium*, and other fungi.

Pammel (24) briefly described a serious root rot and stalk disease of corn and later with King (25) published their studies on a Fusarium disease of corn and sorghum.

This fusarium disease attacks the roots, the stalks, and the ears of corn at least some seasons. It has not been determined whether all the symptoms are caused by the same organism or not.

Under the subject heading "Ear Rot," it is stated that—

the molds are of three kinds. One attacks the kernels, husks, cobs, and sheaths, the threads of the mold occurring thru the cobs and sheaths, and destroying the kernels completely. The second kind of mold produces a deep pink or red color. The threads of this fungus are felty and penetrate the husks. The kernel becomes brittle and red. The third type of mold attacks an occasional kernel and is not so serious. Its threads are white. A fourth fungus, parasitic in character, was found on the ears, stems, and sheaths, and also on the roots. It is known as Diplodia Rot.

Regarding the Fusarium—

the mycelium penetrates not only the living cells, but also occurs in the intercellular spaces. It occurs abundantly in the embryo and endosperm of the seed. The inoculation experiments indicate that the fungus enters not only with the seed, by seminal infection, but that the undeveloped shoots in the axils of the leaves probably are responsible for some of the infection in the field. The disease probably spreads largely with the seed corn.

No determination of the Fusarium was made by the authors. They observed the perithecia of Gibberella abundantly on the sheaths and stem of corn plants but were unable to show that this fungus is connected with the Fusarium studied.

Hoffer and Holbert (18) in 1918 first published on their extensive studies of the root, stalk, and ear rot diseases of corn. It was stated that—

inconspicuous rotting of the stalks, of the ears, and of the roots, may take place with no apparent injury. The kernels from ears borne on diseased plants will have seedling characteristics which can be noted usually on the germinator. Those seedlings which have rotted embryos and stalks indicate the ears to be discarded for seed purposes. The harmful organisms referred to in this bulletin are species of *Gibberella*, *Fusarium*, *Verticillium*, *Rhizopus*, and *Pseudomonas*.

No determination of the species was mentioned at this time. In a later publication (20) it is reported that—

these rot diseases are caused by a number of factors working more or less together, some of which are well known and others less fully known. Investigations have shown that certain fungi *G. acervalis* (Moug) Wr. and *G. saubinetii* (Mont.) Sacc. as well as certain bacteria are commonly present in diseased corn plants in the field. On the germinator those organisms as well as certain molds (*Rhizopus*, *Aspergillus*, etc.) also may occur on diseased, weak, or immature kernels and seedlings.

These authors in a recent abstract (21) on corn root and stalk rots consider the pathogenes are chiefly species of *Fusarium* and *Gibberella*.

The common wheat scab organism, *G. saubinetii* (Mont.) Sacc. is probably the most common pathogene responsible for much of the root and stalk rotting of corn plants in the Central States.

Hoffer (*17*) reports similar observations for sweet corn—

It is believed that the greatest damage to large numbers of seed ears by harmful organisms occurs when the ears are left in the field for long periods after maturity.

Stover (*34*) in Ohio testing seed corn for germination mentions the presence of Fusarium-like fungi. Gilman (*14*) reports a Fusarium-wilt of corn in Iowa. Isolations established the presence of a Fusarium in 93 per cent of the cases with seedlings 6 inches high showing a brown discoloration of the vascular system at the crown.

The literature as reviewed establishes the fact that exclusive of Diplodia, only species of Fusarium have been definitely established with the rot diseases of corn. Germination tests have been observed to reveal the presence of the fungi, but no studies have shown the method by which the pathogenes are carried in what appears to be healthy, normal seed corn.

METHODS

The manner in which infection is carried in seeds showing no external symptoms was determined by germination, cultural, and histological studies.

A representative sample of over 100 kernels was selected from each ear of corn, 10 of which were used for each germination. The germination test was conducted for the most part in a Geneva germinator and with a modified rag-doll type of germinator. This method of germination gives complete evidence as to the relative germination or viability. Such a test does not provide in all cases for an accurate determination of what fungi are being carried internal of the seed. The seed from harvesting to the usual time of germination is exposed to contamination by many saprophytic and in some instances parasitic fungi. The growth from such superficial adhering spores may be so rapid and abundant as to make the identity of the internal parasites impossible. At the end of 7 or 10 days the seedlings were examined for internal symptoms as suggested by Hoffer and Holbert (*20*) in their studies. This point will be discussed in more detail in a later paper on seedling and stalk infection.

The most accurate test that we have found for determining the presence of fungi internal of seed corn, and one which at the same time readily permits of the identification of the fungi, is carried out by disinfecting and planting the kernels or crushed kernels in sterile culture medium in Petri dishes as shown in Plates 3, 4, and 11. Fifteen or more kernels are disinfected in a test tube 150 by 20 mm. for one minute in a solution of 50 per cent alcohol containing 1 gm. of bichlorid of mercury in each liter. This solution is known as a 1 to 1,000 $HgCl_2$ in 50 per cent alcohol. Following this treatment the kernels are washed in the same tube with two successive washings with 20 cc. each of sterile water, and immediately 10 kernels are removed aseptically with sterile forceps and placed with the germ side down on 20 cc. of nutrient dextrose agar in a sterile culture dish. Further, 5 of the remaining kernels are each placed in a sterile culture dish, and with a sterile scalpel the point of the kernel, which is the portion that contains most of the internal infection, is cut off $^1/_8$ to $^1/_5$ inch from the end; then with a strong sterile forcep each point is placed in the mouth of a heavy-walled tube (it requires a strong tube and strong forceps, as crushing is not easy) 150 by 20 mm., containing 10 cc. of sterile nutrient dextrose agar medium at 43° C.; the point is thoroughly crushed and shaken down into the medium,

then well mixed and poured into the sterile culture dish containing the remaining part of the kernel. These methods were used extensively by the senior author in his studies on fungi internal of Flax in 1904 [3] and wheat in 1909 (*28*). In this manner a greater distribution of the mycelium or spores is possible and allows for accurate interpretation in instances where more than one fungus is being carried. Some of the kernels carried three parasitic fungi (Pl. 5, 10, and 11); crushing was the most efficient means of determining all fungi present; on the plate containing uncrushed kernels the faster growing fungi inhibited the others. The plates were read at the end of 7 to 10 days for the presence of all bacterial and fungous growth.

In most of the cultural plate work a dextrose-peptone agar (tap water 1,000 cc., dextrose 10 gm., peptone 1 gm., agar 15 gm.) was of great value in differentiating the several parastic fungi. Twenty cubic centimeters of medium were used in all cultural plates in which 10 kernels of corn were placed for germination. For cultural work in further differentiating the various pathogenes potato starch, rice, and lima bean and sweet potato agar. were used as media. None of the media was standardized because the slight acidity resulting was favorable for differentiating in particular the various species of Fusarium.

Owing to the texture of the ripened kernel, histological methods of investigation meet with difficulties. The germ end and the cob, which are most important for studying, are very horny in texture and difficult to section. Fixation is not so difficult, and in our work Carnoy's killer was found most satisfactory for thorough penetration. If the kernels to be fixed are first cut in half, better killing and infiltration of paraffin is secured. The kernels are best softened for the killer by soaking for 24 hours in warm water, boiling till inhibition is complete, or steaming for 10 minutes in the autoclave. Even this treatment does not give satisfactory softening of the cap, which is the most difficult to cut. After killing, the kernels are dehydrated by the usual method, and serial sections are cut at 10 to 15μ in thickness on the microtome. All material was differentiated with Flemming's triple stain. Rotted kernels resulting from infection with *Diplodia*, *Gibberella*, and *Fusarium moniliforme* are easily prepared for histological studies by soaking eight hours in water.

CEPHALOSPORIUM [4] SACCHARI BUTLER

A fungus unlike any previously reported in this country as far, as we could determine, was found very prevalent internal of seed corn. This fungus morphologically agrees with the description of *Cephalosporium sacchari* Butler, as reported by Butler and Kahn (*8*) on sugar cane in India. Butler (*7*) also found this fungus on sugar canes shipped from the United States to India. In view of these facts and because of the close

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[3] MANNS, Thomas F. FUNGI OF FLAX SICK SOIL AND FLAX SEED. 1904. Unpublished manuscript (Master's thesis) filed in Dept. of Botany, College of Agriculture, Fargo, N. Dak.

[4] In February, 1922 and again in April of the same year, Dr. E. J. Butler, of the Imperial Bureau of Mycology, Kew, England, kindly sent the authors cultures of *Cephalosporium sacchari* isolated from sugar cane by Dr. Shaw, of Pusa, India. There was some question in Dr. Butler's mind as to whether the first isolations were the same as the organism he originally described as *Cephalosporium sacchari*. In the cultures sent in April, 1922, Dr. Butler in examining them stated that "It seems to be pretty near the fungus as I know it." Both of these cultures in our hands proved to be a Fusarium, a fungus entirely different from the organism which we have tentatively referred to as *Cephalosporium sacchari* Butler. This Fusarium sent us by Dr. Butler when grown on nutrient dextrose agar gives a strong purple color. It is probable that the organism we have tentatively referred to the species *Cephalosporium sacchari* is entirely different; if the cultures Dr. Butler sent us are identical with the organism he described as *Cephalosporium sacchari*, then his species should become *Fusarium sacchari* (Butler). Our *Cephalosporium* must receive then further consideration. (This footnote was added February, 1923.)

relationship between the two hosts, it seems better to refer our fungus tentatively to this form than to create confusion by describing a new species. Further studies are in progress to determine the status of our fungus.

Butler and Khan describe the symptoms on half-grown canes as follows:

At this period affected canes lag in growth, and stunted, single stools, or patches of varying size, may soon be observed scattered through the fields in which the disease is prevalent. From this on until the time of harvest withering of individual canes, or even of whole stools occurs. The leaves dry up, as if insufficiently supplied with water, followed by the stems which become light and hollow. If the cane be split longitudinally when the leaves are just observed to wither, a characteristic discoloration of the pith may be observed.

With the exception of a slight yellowing discoloration at the germ end, no external symptoms on seed corn has so far been observed which assist in determining the internal infection by this fungus. Physical characters of the cob, except slight discoloration, have given no indications. Shallow dents, dull appearance, loose cap of kernels have not been correlated with infection. In some instances the pith at the butts of ears is discolored and shredded, but this condition is not more associated with infection by this parasite than with that by any of the other species studied. The infection so far has been determined only in kernels that appear normal. The fungus has been found occasionally associated with the typical kernel rot produced by *Fusarium moniliforme*, but thus far we have not found it responsible for any specific symptom of disease on kernel. Germination of kernels does not appear to be seriously inhibited. On the agar plates the roots are often discolored about the seventh day. Inoculations at nodes and internodes in plants at various stages of development up to maturity has established the parasitic character of this fungus on corn. On the germinator the fungus grows out at the cap and develops a very modified white growth of mycelium. The mycelium is white and does not spread in growth like the other forms. In the limited growth numerous short white filaments are observed. These range from an ⅛ to ¼ inch in height and are coremial-like growths (Pl. 10, G.). Infection with *F. moniliforme* may also be associated with this fungus in which case the more spreading and effused growth overruns and conceals the growth of *Cephalosporium sacchari*.

On culture media the growth is flat with little aerial mycelium produced. In some cultures, upright white coremial strands appear, as shown in Plate 10, G. The oppressed growth on the surface of sugar media as well as the spores en mass are salmon colored. On starch media a slight effused grayish growth is produced. Butler and Khan (8) give the following discussion of spores:

Conidia borne on short, simple or branched, lateral hyphae and also terminally on the ultimate branches of the mycelium. They measure 4–12 μ (usually 5–8 μ) by 2–3 μ when formed, but increase in size prior to germination. Their shape varies from shortly oval to ovoid or long elliptical. Occasionally they are curved or with one side flattened. Some become septate prior to germination, the septa being 1–3 in number.

The conidia as described by these authors are aggregated into globules at the tip of conidiophores. They are typical glomerules, but the spores are not held together in a slime as is characteristic for the genera Hyalopus and Gliobotrys. We find the conidia on the host measure

3.5 to 8 μ by 1.8 to 2.6 μ. On culture media their range is somewhat longer, as 4.3 to 10 μ by 1.7 to 3 μ. The conidiophores range from 16 to 40 μ in length. The conidia at time of germination are much larger and often septate, as shown in Plate 1, B.

GIBBERELLA SAUBINETII[5] (MONT.) SACC

This parasite has been observed extensively on cereals in this country as well as abroad. Johnson, Dickson, and Johann (*22*) have shown this to be the most prevalent organism in producing wheat scab. In their study of over 1,000 specimens collected in 15 states, *Gibberella saubinetii* proved to be the chief causal organism. Hoffer and Holbert (*20*) mention its importance in connection with the root and stalk rot of corn. The description of Fusarium 2 by Burrill and Barrett (*6*) strongly indicates they were working with this species.

As with the preceding species, no uniform symptom is constantly associated with the infection of seed corn by this parasite. Plate 13, A, B, illustrates the extreme type of infection. We have consistently observed such infection confined to the tip of the ear. Such ears show a superficial growth of the mycelium between the rows of kernels which are found to be rotted throughout. In some instances the silk is matted with the mycelium. It is possible that such an association indicates that infection was established before maturity. The ear illustrated in Plate 13, A, was exposed to the changeable weather conditions long after the usual harvest time. Kernels from the tip of this ear were disinfected and crushed in a melted tube of agar for a poured plate. A pure growth of *Gibberella saubinetii* was recovered. Kernels from the butt of this ear treated in a similar manner showed no fungous growth.

The symptoms and effect in the kernels are very similar to that of wheat scab infection. White varieties of corn often show a pink discoloration of the kernels, but it is not so conspicuous on yellow varieties. On shelling the kernels from the infected end, a pinkish discoloration of the fruit cups is often revealed. This appearance is only evident where heavy infection occurs. The rotted kernels are very brittle. The starch in the endosperm is loose or readily crumbles. The embryo in such seeds is completely destroyed. All parts of the kernels under such conditions are invaded by the mycelium. The mycelium is intracellular and intercellular with the same resultant effect on the fruit as reported by Adams (*1*, *2*) for wheat scab infection.

Infection of seed corn is also found which to all external appearance is healthy. On the germinator such corn will show the effused growth of the fungus at the cap in about five days. Germination is retarded and in many instances inhibited on the germinator by the presence of this parasite. The perfect stage has developed on infected kernels that have been allowed to dry out on the germinator under conditions of room temperature. We have not observed the perithecia to develop in any of our cultures. Perithecia are commonly found fruiting at the nodes of old cornstalks in the field. The perfect stage has been found fruiting abundantly on kernels as well as at the butt and in the fruit cups on ears overwintering in the field.

[5] See footnote 4, p. 500.

It is a common observation that where wheat follows corn wheat scab is more prevalent. The ascospores from infected cornstalks, kernels, and cobs overwintering in the field are, no doubt, in part the source of the inoculum. The kernels on the terminal portion of ears appears to be as subject to infection as any of the kernels in wheat heads. This would appear to have been the method of infection of the ears shown in Plate 13, A, B.

Hoffer, Johnson, and Atanasoff (*19*) report *Gibberella saubinetii* from corn and wheat which was used for inoculation on wheat, saying that—

the organisms from both sources have also been found to be similar morphologically. In view of the facts developed by this evidence, it seems certain that these are intercrop parasites which are of great importance in developing control measures for one of the rots of the root, stalk, and ear of corn and for scab of wheat.

With the usual abundant fruiting of *Gibberella saubinetii* on old cornstalks it is not difficult to secure pure cultures in order to determine the species isolated from the growth on germinating kernels. The fungus produces a very effused growth on the various media used. When the surface of the medium is completely covered by the mycelium a carmine red appearance is produced and the aerial mycelium becomes yellowish in color. Conidia which are 3-septate to 5-septate developed very sparingly in culture, and no true chlamdospores were found. The conidia observed in our cultures were for the greater part 5-septate. Measurements were made from conidia on dextrose agar and averaged 42 to 52.5 μ by 3.5 to 4.7 μ. A good detailed description of this species is given by Wollenweber (*40*).

FUSARIUM MONILIFORME SHELDON

This fungus, described by Sheldon (*29*) in 1904 was not referred to again until the association of this species with seedling-blight of conifers. Spaulding (*32*) mentions this species among some damping-off fungi on seedling conifers. Hartley, Merrill, and Rhoads (*15*) found it to be the most virulent of those tested on seedling conifers. As previously mentioned, Valleau (*36*) considers this fungus as the common cause of the root, stalk, and ear rot of corn. Norton and Chen (*23*) recently called attention to a new parasite of corn. They discussed the resemblance of their fungus to *Oospora verticilloides* Sacc. They consider their fungus is similar to *Fusarium moniliforme*, in so far as the type of catenulate conidia is concerned, but failed to find any fusiform or macroconidia as described by Sheldon (*29*). No mention of macroconidia since Sheldon's studies has been described by any investigators in their studies with this fungus. They are sparingly developed, which accounts for their not being commonly observed. It is of further interest that a species of Fusarium should possess catenulate conidia, as this does not agree with the characters of this form genus. Appel and Wollenweber (*3*) and Sherbakoff (*30*) do not consider catenulate conidia in their description of this form genus.

The fungus *Oospora verticilloides* described by Saccardo (*27*) on corn is no doubt identical with *Fusarium moniliforme*. The illustration of spores and their method of formation and measurement (*26*) are identical. Later Deckenbach (*9, 10*) and Tiraboschi (*35*) observed this fungus on corn in connection with their studies on pellegra. Deckenbach (*9*) observed the violet and lavender color this fungus takes on under cul-

tural condition. He also reports finding a similar color on seed corn naturally sick with Oospora. This discoloration of the grains we have commonly observed with infected kernels overwintering under field conditions. Tiraboschi (*35*) gives more of a detailed study to this fungus, and his results and illustrations are very convincing. The confusion in the past regarding the identity of *Fusarium moniliforme* and *Oospora verticilloides* is no doubt the result of the macronconidia being overlooked.

The symptoms on seed corn produced by this fungus are numerous and more commonly found than those produced by any of the other internal parasites. The first conspicuous symptom is that of the typical kernel rot. Such infected kernels appear irregularly distributed in the ear as shown in Plate 12, D. This kernel rot in appearance agrees with the one described by Burrill and Barret (*6*) as caused by their Fusarium 3.

The condition of such infected kernels is similar to Sheldon's (*29*) description. The kernels appear slightly shrunken and light brown in color. Such kernels are soft and fragile, with the contents powdery, except for the embryos, which are discolored and shrunken. In some instances a slight development of mycelium and spores is found on the surface. Other symptoms of infected kernels are discolorations of the seed coat. These discolorations vary from a light brown to pink and lavender in color. They may be evident on any part of the kernel but are most commonly found near the germ end. This discoloration is the result of the development of mycelium between the integuments. These symptoms become more pronounced during germination because of the activity of the fungus. In some instances the fungus may spread from the germ end under the pericarp so as to discolor the lower half of the kernel.

Several samples of bin-selected ears showed cracking of kernels just above the cap, as illustrated in Plate 12, B. This cracking would involve as many as 8 or 10 adjacent kernels in a row. Such symptoms were found irregularly distributed throughout the ear. In the majority of such cases *Fusarium moniliforme* was found associated with such injury. The primary cause of this condition was not determined. The influence of excessive moisture and growth may have been the contributing factors. However, it would appear from all evidence that the fungus became established subsequent to the cracking. The embryo in these cracked kernels is frequently killed by the fungus. However, such seeds seldom develop into strong seedlings, owing to the amount of infection. A series of 20 cracked and 20 normal-appearing kernels from the same ear were planted in the greenhouse. Only 9 of the cracked kernels developed, whereas perfect germination and development were secured with the ones not cracked. During germination, when the plants are 6 to 8 inches high, it is found on removing the seedling that the seed coat is conspicuously discolored. The pericarp takes on a deep lavender color.

On the germinator, infection first appears as a slight effused growth around the cap. As a rule, the fungus ramifies between the pericarp and epidermis, resulting in pronounced discoloration of the pericarp and involving the lower half of the kernel. Often the discoloration will appear as irregular streaks, extending from the germ end toward the crown. The germination of corn is inhibited by this fungus but not as severely as was observed with *Gibberella saubinetii* and *Diplodia zeae*.

The kernel-rotted seeds (Pl. 12, E) show extensive ramification of the fungus. All parts of the kernels are invaded by the mycelium, which is intercellular as well as intracellular. The resultant effect is similar to the rot produced by *Gibberella saubinetii* on corn and wheat.

On culture media this fungus makes a slight, to dense effused growth. With some strains there is a conspicuous pigmentation of the vegetative growth. The dense growth of mycelium often appears lavender in color, although this was not observed to be consistent for any one strain regardless of the medium. The catenulate conidia can easily be determined by examining the aerial growth with the low-power objective. The chains of spores vary in number, and the maximum counted was 30 spores in a single chain. It is not uncommon to find the conidia aggregated into heads, as shown in Plate 2, G-H. The conidiophores are simple or branched as shown in Plate 2, D-F. Strands of mycelium with numerous conidiophores often become interlaced. The conidia in chains are slightly pyriform, and the attenuated end of the spore is often sharply muticate. At time of germination they are considerably larger and often with one or two cross septa (Pl. 2, B). The following measurements of microconidia by various investigators are given for comparison:

Saccardo (*27*), host material	8 to 10μ by 2.5 to 3μ.
Sheldon (*29*)	6 to 10μ length.
Tiraboschi (*35*)	5 to 7μ by 2 to 3.5μ.
Hartley, Merrill, and Rhoads (*15*)	Prune agar 4.8 to 6.3μ by 2.2 to 3.1μ; corn meal 7.4 to 11.1μ length.
Authors, host material	7 to 9μ by 2.6 to 3.5μ.
Authors, dextrose medium	6.7 to 12μ by 2.5 to 3.5μ.

The macroconidia are sparingly developed and were first observed in cultures that were at least 2 months old. Some strains even as old as that failed to develop macroconidia. We did not observe any of the macroconidia on host material. Marcoconidia in our cultures were 3-septate and measured 19 to 31.5 μ by 2.5 to 3.5 μ. Cultures on rice agar were first found to produce macroconidia, and similar results were obtained with cultures on steamed corn meal.

DIPLODIA ZEAE (SCHW.) LEV.

The term "dry rot" is often used in describing the effects on the ears produced by this fungus. However, the term seems no more applicable in this case than for the kernel-rot produced by *Fusarium moniliforme* and *Gibberella saubinetii*.

In extreme infection of the ears the matting and rotting are similar to that described for *Gibberella saubinetii* (Pl. 13) and which by growers is called moldy ears. The mycelium is established in a like manner, but no pink coloration is produced, such as is often found with infection of *G. saubinetii*. Kernels of both white and yellow varieties of corn show considerable blackening, which varies from irregular streaks to large areas involving more or less the entire kernel. In some instances the development of pycnidia is so abundant as to produce this blackened appearance. It is not uncommon to find scattered infected kernels at the tip of ears of which the upper part is brown to black in color. The pericarp in this region is of a fragile texture, and inside is found a powdery brown mass consisting of corroded starch grains with abundant brown septate mycelium.

A study of diseased kernels in cross section has shown the fungus well established between the aleurone layer and integuments. Immature pycnidia have been observed in this region. The mycelium ramifies through the endosperm tissue. The starch grains appear corroded, as reported by Heald, Wilcox, and Pool (*16*). Rotted kernels present the same physical condition as that described for *Fusarium moniliforme*.

Kernels which appear normal but show infection during germination often produce black streaks at the germ end. This appearance results from developing and progressing of the fungus under the pericarp. An interlaced mass of brown mycelium is often found developing under the pericarp. The most conspicuous sign is found during germination with the effused growth of white, cottony mycelium at the germ end. Germination is greatly retarded and often inhibited as a result of infection. A summary of extensive germination tests shows that this fungus is more inhibitive to germination than the other parasites described.

The white, cottony effused growth of this fungus in agar cultures is easily determined. The mycelium on the substratum takes on a brownish appearance with age. On rice agar it fruits abundantly. The conidia (pycnospores) are 2-celled and brown in color. Measurements of conidia from infected roots and cultures are the same. Conidia from rice media measure 16.5 to 31.5 μ by 5 to 6 μ.

PREVALENCE

In making a survey of fungi internal of corn in Delaware an appeal was made to several hundred farmers, including many members of the State Corn Growers' Association, requesting at least four ears of seed corn, two of which should represent the best seed corn grown and two ears much above the average from bin selection; the object of making a request of this nature was to secure material from which first-hand information could be obtained on the amount of internal infection carried in seed corn and to learn whether field selection was superior to bin selection. In order to make a comparison with other States a request for cooperation was sent to pathologists and agronomists of 20 States of the corn belt.

A careful review of Table I indicates that there are at least four species of fungi commonly found internal of seed corn which are widely distributed. We fully realize that the survey if not extensive enough to show the condition prevailing in seed corn in the several districts throughout the United States. Superficially the survey shows that the organisms vary in prevalence considerably throughout the different sections. *Cephalosporium sacchari*, *Fusarium moniliforme*, and *Diplodia zeae* seem to have a wide range but are probably more prevalent through the middle tier of States and the southern States, whereas *Gibberella saubinetii* appears more prevalent through the States producing winter wheat.

In North Dakota, from which 25 samples were received out of which 375 kernels were cultured, no *Cephalosporium sacchari* or *Gibberella saubinetii* was found and only a trace of *Fusarium moniliforme* and *Diplodia zeae*. The samples from several or the States were not representative farm samples but were sent as special selections from experiment stations.

TABLE I.—*Survey by States of parasitic fungi internal of seed corn*

State.	Number of samples.	Number of kernels cultured.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Germination.		
							Strong.	Weak.	Dead.
			Per cent.	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*	*Per cent.*
Del.	219	3,285	39.54	5.95	19.92	5.69	86.35	9.56	4.09
Ark.	15	225	22.00	1.33	35.33	3.33	66.00	26.66	7.34
Conn.	12	180	16.66	25.83	41.66	....	84.16	14.16	1.68
Ill.	25	375	2.00	....	12.40	7.20	82.80	16.00	1.20
Ind.	17	255	4.70	4.70	....	....	96.44	3.56	....
Ky.	34	510	3.82	4.11	17.94	4.85	59.70	15.59	24.71
Kans.	12	180	11.66	....	30.00	....	88.33	11.67	....
La.	1	15	....	....	80.00	....	100.00	....	....
Mass.	5	75	....	12.00	10.00	8.00	85.00	6.00	9.00
Md.	10	150	46.00	7.00	12.00	14.00	65.00	35.00	....
Minn.	10	150	10.00	12.00	43.00	....	37.00	55.00	8.00
Miss.	16	240	38.13	....	40.00	3.12	74.37	25.00	.63
Nebr.	14	215	22.85	....	20.00	1.42	85.71	4.28	10.01
N. C.	10	150	38.00	2.00	48.00	....	86.00	14.00	....
N. Dak.	25	375	....	....	.40	.80	85.60	1.20	13.20
N. J.	10	150	24.00	21.00	17.00	2.00	79.00	21.00	....
N. Y.	6	90	....	3.33	....	....	96.67	3.33	....
Ohio.	11	165	10.90	22.72	1.82	12.73	49.09	50.91	....
Pa.	14	210	5.71	7.87	....	....	89.57	3.57	6.86
Tex.	7	105	14.28	....	71.42	2.85	21.42	78.58	....
Wis.	7	105	20.00	2.85	....	10.85	97.14	....	2.86

Of the several parasitic fungi carrying internal of seed corn in Delaware *Cephalosporium sacchari* is most prevalent, occurring in the several thousand kernels cultured to the extent of 39.54 per cent. This fungus is frequently found associated in the same kernel with the other parasitic fungi *Fusarium moniliforme, Gibberella saubinetii, and Diplodia zeae.*

Fusarium moniliforme as an internal parasite is found to the extent of 19.92 per cent in seed corn in Delaware. Its latitude or range of infection is the same as that of *Cephalosporium sacchari,* and frequently both are found occupying the same kernels. The fact that *C. sacchari* and *F. moniliforme* fruit abundantly both internally and externally of seed corn would indicate that surface contamination must be very common.

Gibberella saubinetii is much less common as an internal parasite of corn in Delaware and throughout the United States than either *Cephalosporium sacchari* or *Fusarium moniliforme.* In Delaware 5.95 per cent of the kernels cultured gave this fungus. The fact that *G. saubinetii* fruits more sparingly as an ear rot and kernel infection of corn and produces no chlamydospores would indicate that as a seed-carrying organism its possibility of distribution is much more limited than that of either *C. sacchari* or *Fusarium moniliforme.* Its prevalence seems to vary greatly in the several districts.

Diplodia zeae like the other parasites considered above may be internal of seed corn that appears entirely healthy. Its prevalence in Delaware seed corn for 1920 was 5.69 per cent. As a parasite its range of activity, judging from internal infection, is identical with that of *Cephalosporium sacchari* and *Fusarium moniliforme*—probably not quite so great in the Gulf States as in the Central States.

Species of the following fungi in several instances also have been found internal of seed corn: Aspergillus, Penicillium, Cladosporium, Alternaria, Rhizopus, Torula, Hormodendron, Chaetomium, Helminthosporium, and Spicaria. *Colletotrichum cereale* has also been found. Adams and Russell (2) have shown *Rhizopus nigricans* to be a parasite of the storage tissue of the scutellum and a limiting factor in germination. The junior writer has also noted the same condition for species of Penicillium and Aspergillus. It is possible the other forms are likewise concerned with inhibiting germination. Unlike the four species described above, these fungi are not so consistently found associated with seed corn, hence are of much less importance. Also several times we have met with two species of bacteria, a yellow organism and a white organism; the importance of these we have not determined. The extent of these associated fungi and bacteria as internal organisms is given in detail in the Table II, which shows the general distribution and prevalence of fungi internal of seed corn by samples from the several States.

By a careful study of Table II it is occasionally seen that there are samples of corn running low in germination that contain no internal parasites. The explanation of this may be root rot, so weakening the plants as to effect germination, or possibly storage difficulties which reduced viability. As a rule, weakened or impaired germination seems to be associated with internal infection, the most inhibiting parasites being *Diplodia zeae* and *Gibberella saubinetii.*

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	DELAWARE							
		Per ct.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
1A	Yellow Dent, local	60		20			90	10
1B	.....do						100	
1C	.....do						100	
1D	.....do	40					100	
2A	Boone County White	100					90	10
2B	.....do	20					80	10
2C	.....do						100	
2D	.....do		20				90	
3A	Johnson County White	20		25		[10] 30	100	
3B	.....do	100			20		100	
3C	.....do	100					100	
3D	.....do	100					100	
4A	Yellow Dent, local	100					80	
4B	.....do	80		20			100	
4C	.....do	100		50			100	
4D	.....do	100		80			100	
5A	White Dent, local						100	
5B	.....do	60					100	
6A	.....do						100	
6B	....do	100					100	
7A	Reid Yellow Dent				60		100	
7B	.....do				80		70	30

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	DELAWARE—continued	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.
8A	Reid Yellow Dent				60	[10] 40	100	
8B	do	100			40		70	30
9A	Yellow Dent, local	20		20	20		100	
9B	do	60		60			100	
10A	do	100	40				100	
10B	do	80	20		70		30	
11A	Early Bristol				20		100	
11B	do	80		40			100	
12A	do	100		40			100	
12B	do	100		40			100	
13A	White Dent, local	60					100	
13B	do	100		20			100	
14A	do						90	
14B	do	100					100	
15A	Yellow Dent, local	80					100	
15B	do						100	
15C	Yellow Dent, local	80					100	
15D	do	20				[10] 20	100	
16A	White Capped Yellow Dent, local.	20					100	
16B	do	100	40				100	
17A	do	60		40			100	
17B	do	100			10		70	30
18A	do	80		20			100	
18B	do						100	
18C	do	80		20			100	
18D	do	40		20	20		100	
19A	White Dent, local			20			100	
19B	do	60	20				100	
19C	do	100	60	60			80	
19D	do	40					100	
20	Yellow Dent, local			100			80	
21A	do	100					100	
21B	do			80			100	
21C	do	100					70	30
21D	do	60		60			100	
21E	do	100					70	30
22A	do						100	
22B	do	100				[7] 60	100	
22C	do		20			[3] 50	72	
23A	White capped Yellow Dent, local						100	
23B	do						90	10
24A	do		40		20		10	70
24B	do	60					100	
25A	Johnson County White	60					10	90
25B	do	20					90	
25C	do						100	
25D	do	20					100	
26A	Reid Yellow Dent	20					100	
26B	do	20					90	

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae*[?] *forme.*	Other fungi.[b]	Germination. Strong.	Germination. Weak.
	DELAWARE—continued	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
27A	Reid Yellow Dent					[3] 20	100	
27B	.....do					[7] 60	60	30
28A	Yellow Dent, local	100		30			100	
28B	.....do	100		20			100	
28C	.....do	80		20			100	
28D	.....do	20		100	40		90	
29A	Reid Yellow Dent	30		100			100	
29B	.....do	100					100	
30A	.....do		100				100	
30B	.....do	100	100				100	
31A	White Dent, local			80			50	
31B	.....do		25				70	30
31C	.....do	100		40			100	
31D	.....do	60	20	10			60	30
32A	.....do	100	40	100			90	
32B	.....do	30		30	20		100	
32C	.....do	100	40	10			90	
32D	.....do	100	100	20			100	
33A	.....do	60		40	60			40
33B	.....do	80	70	20			100	
33C	.....do						100	
33D	.....do	60			80			40
33E	.....do	100		60			100	
34A	Reid Yellow Dent	60		20			80	
34B	.....do			80			80	
34C	.....do			80			80	20
34D	.....do			100			40	40
34E	.....do	60		100			60	30
35A	White Capped Yellow Dent, local			20	10		50	30
35B	.....do	20					100	
35C	.....do		40				60	40
35D	.....do		40				100	
36A	.....do		10	20			30	50
36B	.....do						10	90
37A	.....do						100	
37B	.....do						100	
38A	.....do						80	20
38B	.....do	50		88	20		100	
38C	.....do			60			100	
39A	.....do	60					70	30
39B	.....do	40					100	
40A	White Dent, local	20					100	
40B	.....do	40					100	
40C	.....do						100	
40D	.....do	20					100	
40E	.....do	60		40			100	
41A	.....do	20		10	10		90	
41B	.....do		40		10		70	
41C	.....do	20	40				90	
42A	Yellow Dent, local	40		40	60		50	
42B	.....do	100					70	30

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Clados porium* spp.; 3, *Peinicillum* spp.; 4, *Allernaria* spp.; 5, *Helminthos porium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No. [a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.	Germination.	
							Strong.	Weak.
	DELAWARE—continued							
		Per ct.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
43A	White Capped Yellow Dent, local	......	......	......	60	......	100	......
43B	.....do	......	......	......	......	......	40	60
43C	.....do	......	......	......	......	......	100	......
43D	.....do	20	......	......	......	......	100	......
44A	Yellow Dent, local	80	......	40	......	......	90	......
44B	.....do	40	20	......	......	......	100	......
45A	.....do	100	......	20	......	......	100	......
45B	.....do	......	50	40	......	......	100	......
46A	Johnson County White	......	......	......	......	......	100	......
46B	.....do	......	......	......	......	......	100	......
46C	.....do	......	......	......	......	......	100	......
46D	.....do	......	......	......	......	......	100	......
46E	.....do	......	......	......	......	......	100	......
46F	.....do	......	......	......	......	......	100	......
47A	Cloud's Yellow Dent	......	20	......	......	......	90	......
47B	.....do	......	......	......	......	......	100	......
47C	.....do	......	......	......	......	......	100	......
47D	.....do	......	......	......	......	......	100	......
48A	Yellow Dent, local	......	......	......	......	......	100	......
48B	.....do	......	......	......	......	......	40	40
48C	.....do	......	......	40	......	......	90	10
48D	.....do	20	......	40	......	......	100	......
49A	White Dent, local	60	......	60	......	......	100	......
49B	.....do	60	......	60	......	......	90	......
49C	.....do	100	......	100	......	......	100	......
49D	.....do	100	......	......	......	......	100	......
50	Illinois Low Oil	60	......	20	......	......	70	......
51	White Dent, local	100	......	75	25	......	100	......
52	.....do	100	......	75	40	......	40	30
53	.....do	25	......	100	......	......	100	......
54	.....do	60	......	60	40	......	20	80
55	.....do	......	......	......	40	......	40	......
56	.....do	30	......	66	......	......	100	......
57	.....do	50	......	......	10	......	100	......
58	.....do	100	......	50	40	......	40	60
59	.....do	40	......	40	......	......	90	10
60	.....do	60	......	40	......	......	100	......
61	.....do	100	......	......	......	......	100	......
62	.....do	100	......	20	......	......	100	......
63	.....do	80	......	80	......	......	100	......
64	.....do	100	......	......	......	......	100	......
65	.....do	100	......	100	......	......	60	40
66	.....do	100	......	......	......	......	100	......
67	.....do	......	......	100	......	......	30	70
68	.....do	......	......	......	100	......	100	......
69	.....do	......	......	25	......	......	100	......
70	.....do	......	......	......	......	......	100	......
71	.....do	20	......	50	40	......	80	20
72	.....do	100	......	......	......	......	70	30
73	.....do	......	......	100	......	......	100	......

[a] Letters A, B, C, D, etc., indicate different ears from same source.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	DELAWARE—continued	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.
74	White Dent, local					[1]10	100	
75A	do	40					100	
75B	do	10		20			100	
75C	do	40		40			100	
75D	do	60		10		[3]40	80	10
76A	Yellow Dent, local		40	20			90	10
76B	do			20		[3]100	100	
76C	do						100	
77	White Dent, local						100	
78A	do	20			20		100	
78B	do	20					100	
78C	do	40					100	
78D	do	100				[12]100	100	
79A	Yellow Dent. local	20		100			10	90
79B	do	20				[9]40	40	60
79C	do	100					60	40
79D	do						100	
80A	Golden Beauty		80				70	30
80B	do							100
80C	do				20		60	40
80D	do					[3]20	100	
80E	do					[11]20 [12]20	100	
80F	do						100	
80G	do						100	
80H	do				20		60	40
81A	White Dent, local			20			40	60
81B	do			100			70	30
81C	do		80	80			20	80
81D	do	20		100			30	70
81E	do	40				[3]60	50	50
82A	Bristol Dent	100					100	
82B	do	40					100	
82C	do	100					100	
	ARKANSAS							
83	Washington County	20		60			70	30
84	Paymaster	40		10			80	20
85	Whartley's Prolific	30		20			90	
86	Hackberry	20	20	100			10	90
87	McFarland			20			30	70
88	Southern Beauty			40			60	40
89	Chesholm	60		40	10		30	70
90	U. S. 165				10		50	50
91	Stewarts Yellow Dent	20		60			100	
92	U. S. Sel. No. 77			20		[1]20		
93	Experiment Station Yellow			40			100	
94	Boone County White	100		80			90	10
95	Laguna Mexican June	20		40	10		90	10
96	Biggs-Seven-Ear				20		100	
97	Eureka	20					90	10

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubientii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	CONNECTICUT							
		Per ct.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
98	Bearsley's Leaming						100	
99	Webber's Dent		50				30	70
100	Early Lakeside		10			[4] 30		80
101	King Philip		10				100	
102	Sharon White Cap						100	
103	Pride of North		70				100	
104	Golden Nugget	20	10				100	
105	Sanford White		50				100	
106	Longfellow		60					100
107	Rhode Island White		20	40			100	
108	Century Dent			10			100	
109	Twelve Row		30				100	
	ILLINOIS							
110	Western Plowman						70	30
111	Reid Yellow Dent						100	
112	.....do			40	20	[3] 20	100	
113	.....do				20		70	30
114	.....do				20		60	40
115	.....do					[4] 10	60	40
116	Leaming			20			30	70
117	Boone County White						100	
118	Silvermine						100	
119	Champion White Pearl			20			20	80
120	Illinois High Oil						100	
121	Illinois Low Oil						100	
122	Illinois High Protein			20	20		100	
123	Illinois Low Protein				40		60	40
124	Yellow Dent			40	40		60	40
125	White Dent	30		10			100	
126	Variety not given			40			100	
127	.....do			60		[12] 20	90	10
128	.....do						100	
129	.....do						100	
130	Yellow Dent				20		80	
131	.....do					[10] 20	100	
132	.....do			60			80	20
133	Variety not given	20					90	
134	.....do						100	
135	Colclessons White Dent			20			100	
	INDIANA							
136	Johnson County Yellow Dent						100	
137	Johnson County White Dent					[11] 20	100	
138	Reid Yellow Dent						100	
139	Vogliss White Dent			20		[4] 20	100	
140	Reid Yellow Dent						100	
141	Johnson County White Dent						100	
142	.....do						100	
143	Colclessons White Dent	80					80	20

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	INDIANA—continued	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
144	Reid Yellow Dent	......	20	......	......	......	100	......
145	Johnson County White Dent	......	60	......	......	......	60	40
146	Voglers White Dent	......	......	......	......	......	100	......
147	Yellow	......	......	......	......	......	100	......
148	Flesh Corn	......	......	......	......	......	100	......
149	Yellow	......	......	......	......	......	100	......
150	White	......	......	......	......	......	100	......
151	Yellow	......	......	......	......	......	100	......
	KANSAS							
152	Pride of Salem	......	......	......	......	......	100	......
153	.....do	......	......	......	......	......	100	......
154	.....do	......	......	60	......	......	100	......
155	.....do	......	......	60	......	......	100	......
156	.....do	40	......	40	......	......	70	30
157	.....do	......	......	20	......	......	90	10
158	.....do	......	......	60	......	......	70	30
159	.....do	20	......	20	......	......	90	10
160	.....do	80	......	40	......	......	80	20
161	.....do	......	......	......	......	......	100	......
162	.....do	......	......	60	......	......	60	40
163	.....do	......	......	......	......	......	100	......
	KENTUCKY							
164	Variety not given	......	......	......	......	......	100	......
165	.....do	......	......	......	......	......	100	......
166	.....do	......	100	......	......	......	60	40
167	.....do	......	......	20	10	......	60	40
168	.....do	......	......	40	......	......	100	......
169	.....do	......	......	40	......	......	60	40
170	.....do	......	......	10	......	......	90	10
171	.....do	......	......	20	......	......	80	20
172	.....do	20	......	10	10	......	40	60
173	.....do	......	......	10	20	......	30	70
174	.....do	......	......	40	40	......	40	60
175	.....do	......	......	20	40	......	100	......
176	.....do	......	......	......	......	......	100	......
177	.....do	20	......	20	......	......	90	......
178	.....do	......	......	10	10	......	90	......
179	.....do	......	10	40	......	[3] 10	80	......
180	.....do	......	......	100	......	......	40	60
181	.....do	20	......	......	......	......	100	......
182	.....do	40	......	......	......	......	90	10
183	.....do	......	......	......	......	......	100	......
184	.....do	......	......	10	......	......	100	......
185	.....do	20	......	......	......	......	10	90
186	.....do	......	......	30	......	......	70	30
187	.....do	20	......	20	20	......	100	......
188	.....do	......	......	......	10	......	100	......
189	.....do	......	......	......	......	......		

[a] Letters A, B, C, D, etc., indicate different ears from same source.
[b] The small numbers in this column refer to the following numbered fungi: 1 *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	KENTUCKY—continued	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
190	Variety not given	......	......	60	......	[1] 100	......	......
191	.....do	......	......	......	......	[1] 1, 30	......	......
192	.....do	......	......	......	......	[1] 1, 20	......	......
193	.....do	......	......	70	......	[1] 100	......	......
194	.....do	10	10	40	......	[1] 100	......	......
195	.....do	......	......	10	......	[1] 100	......	......
196	.....do	......	......	......	5	......	......	......
197	.....do	......	......	......	......	[1] 10	......	......
	LOUISIANA							
198	Calhound White	......	......	80	......	......	100	......
	MARYLAND							
199	Yellow Dent, local	100	......	......	......	......	80	30
200	.....do	40	......	......	......	......	100	......
201	.....do	......	......	......	......	......	100	......
202	.....do	......	......	......	......	......	100	......
203	.....do	20	......	......	......	......	100	......
204	.....do	60	......	40	60	......	50	50
205	.....do	80	70	20	......	......	100	......
206	.....do	......	......	......	......	......	20	80
207	.....do	60	......	......	80	......	50	50
208	.....do	100	......	60	......	......	50	50
	MASSACHUSETTS							
209	Sweet Corn	......	20	......	20	[4] 30	70	
210	Yellow	......	......	20	......	......	80	20
211	Boone County White	......	......	20	20	......	90	10
212	White Dent	......	......	......	......	......	100	......
213	Yellow Dent	......	40	10	......	[4] 10	80	......
	MINNESOTA							
214	Variety not given	......	......	100	......	[3] 60	......	100
215	.....do	60	......	50	......	......	100	......
216	.....do	......	......	......	......	[3] 100	......	20
217	.....do	......	80	20	......	......	100	......
218	.....do	......	......	50	......	......	50	50
219	.....do	......	40	30	......	[11] 60	20	80
220	.....do	......	......	100	......	......	20	80
221	.....do	......	......	20	......	[3] 100	40	60
222	.....do	40	......	20	......	......	20	80
223	.....do	......	......	40	......	[11] 40	20	80
	MISSISSIPPI							
224	Paymaster (Harpeth)	10	......	40	......	......	60	40
225	Williamson (Cokers)	60	......	60	......	......	100	......
226	Ellis (Cokers)	40	......	60	......	......	60	40

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TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	MISSISSIPPI—continued							
		Per ct.	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*	*Per ct.*
227	Ewing's Mosbys (Station)	60	......	40	......	......	100	......
228	Mosbys (Cokers)	40	......	60	......	......	70	30
229	Hastings	......	......	10	10	......	80	10
230	Station Laguna	60	......	80	......	......	50	50
231	Chesholm (Ferg.)	40	......	40	......	......	100	
232	Marlboro (Wann)	40	......	40	......	......	90	10
233	Paymaster (Neals)	10	......	70	10	......	30	70
234	North Carolina Prolific	10	......	60	10	......	40	60
235	Station Tennessee Red Cob	20	......	......	......	......	80	20
236	Station Tennessee Red Cob 72	100	......	40	......	......	60	40
237	Station Florida Flint	100	......	......	......	......	90	10
238	Whatley's Prolific	20	......	40	20	......	90	10
239	Reid Yellow Dent	......	......	......	......	......	90	10
	NEBRASKA							
240	Yellow Dent, local	80	......	20	......	......	80	20
241	.....do	......	......	......	......	......	100	......
242	.....do	......	......	40	......	......	90	......
243	.....do	......	......	40	......	......	70	30
244	.....do	40	......	20	......	......	100	......
245	.....do	40	......	40	......	......	100	......
246	White Dent, local	......	......	......	......	......	......	......
247	Haynes Yellow Dent	20	......	20	......	......	90	......
248	Yellow Dent, local	......	......	......	......	......	100	......
249	.....do	40	......	20	......	......	80	......
250	.....do	......	......	......	......	[11] 20	100	......
251	Calico Dent, local	80	......	60	......	......	100	......
252	White Cap Dent, local	......	......	......	......	[10] 20	100	......
253	Nebraska White Prize	20	......	20	20	......	90	10
	NEW JERSEY							
254	Variety not given	......	......	......	......	......	50	50
255	.....do	......	60	......	......	......	90	10
256	.....do	......	10	10	......	......	100	......
257	.....do	......	60	20	......	......	20	80
258	.....do	20	20	......	......	......	80	20
259	.....do	100	20	20	......	[4] 20	80	20
260	.....do	......	......	......	......	......	100	......
261	.....do	100	20	20	......	......	90	10
262	.....do	......	20	100	20	[4] 20	80	20
263	.....do	20	......	......	......	......	100	......
	NEW YORK							
264	Variety not given	......	......	......	......	......	100	......
265	.....do	......	......	......	......	......	100	......
266	.....do	......	......	......	......	......	100	......
267	.....do	......	......	......	......	......	100	......
268	.....do	......	20	......	......	......	80	20
269	.....do	......	......	......	......	......	100	......

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[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination.	
							Strong.	Weak.
	NORTH CAROLINA	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.	Per ct.
270	Neals Paymaster			80			20	80
271	Deatons Two Ear	20		60			100	
272	Cooke's Prolific			40			100	
273	Long Valley White	60		40			40	60
274	Grampian						100	
275	Weekleys Improved	60		100			100	
276	Biggs Seven Ear	80		20			100	
277	Horse-Tooth	60	20	40			100	
278	Kickers Indian-Chief			60			100	
279	Lathanis Double	100		40			100	
	NORTH DAKOTA							
280	Variety not given						100	
281	.....do			10			100	
282	.....do						100	
283	.....do						100	
284	.....do						100	
285	.....do						100	
286	.....do				20		80	
287	.....do						100	
288	Minnesota No. 13						100	
289	.....do						100	
290	.....do						100	
291	.....do							( )
292	A Yellow Capped Red							(c)
293	Northwestern Dent					[8] 100	[11] 100	(c)
294	.....do						100	
295	.....do					[12] 20	100	
296	White Rustler						100	
297	.....do						100	
298	.....do						100	
299	Yellow Dent						100	
300	.....do						100	
301	Labels lost in shipment						100	
302	.....do						70	30
303	.....do						100	
304	.....do				10		90	
	OHIO							
305	White Capped Red		70				70	30
306	.....do	20			100			100
307	.....do		20					100
308	Bloody Butcher		40				100	
309	Yellow Dent, local				40		70	30
310	White Cap						100	
311	Snures White Cap		100				30	70
312	Stauffers Yellow Cap		20				100	
313	J. S. Leaming	100		20			30	70
314	Clarage					[3] 20, [12] 20	20	80
315	Stauffers Yellow						20	80

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[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

[c] Probably all frosted; dead.

TABLE II.—*Distribution and prevalence of fungi internal of seed corn*—Continued

No.[a]	State and variety.	*Cephalosporium sacchari.*	*Gibberella saubinetii.*	*Fusarium moniliforme.*	*Diplodia zeae.*	Other fungi.[b]	Germination. Strong.	Germination. Weak.
	PENNSYLVANIA							
316	Johnson County White	40	......	......	......	......	100	......
317	Landis White	......	10	......	......	[1] 10; [3] 20	90	10
318	Reid Yellow Dent	20	......	......	......	......	90	10
319	College White Cap	......	......	......	......	......	100	......
320	Greene County Yellow Dent	20	......	......	......	......	100	......
321	Clouds Yellow Dent	......	100	......	......	......	......	20
322	Golden Yellow Dent	......	......	......	......	......	100	......
323	.....do	......	......	......	......	......	100	......
324	.....do	......	......	......	......	......	90	10
325	.....do	......	......	......	......	[3] 90	80	......
326	Lags Clapper	......	......	......	......	......	100	......
327	Johnson County White	......	......	......	......	......	100	......
328	.....do	......	......	......	......	......	100	......
329	.....do	......	......	......	......	......	100	......
	TEXAS							
330	Johnson County White	60	......	50	......	[1] 20; [3] 20	20	80
331	Chesholm	......	......	40	20	[1, 3] 60	20	80
332	Sure Cropper	......	......	100	......	[1] 80	......	[6] 100
333	.....do	20	......	60	......	[3] 60	60	40
334	.....do	20	......	70	......	[1, 3] 40	......	100
335	.....do	......	......	80	......	[1, 3] 75	20	80
336	.....do	......	......	100	......	[1] 40	30	70
	WISCONSIN							
337	Variety not given	......	......	......	......	......	100	......
338	.....do	100	......	......	......	......	100	......
339	.....do	......	......	......	......	......	100	......
340	.....do	......	......	......	......	......	100	......
341	.....do	......	......	......	......	......	100	......
342	.....do	......	......	......	16	......	100	......
343	.....do	40	20	......	60	......	80	......

[a] Letters A, B, C, D, etc., indicate different ears from same source.

[b] The small numbers in this column refer to the following numbered fungi: 1, *Aspergillus* spp.; 2, *Cladosporium* spp.; 3, *Penicillium* spp.; 4, *Alternaria* spp.; 5, *Helminthosporium* spp.; 6, *Rhizopus* spp.; 7, *Spicaria* spp.; 8, *Hormodendron* spp.; 9, *Torula* spp.; 10, *Chaetomium* spp.; 11, *Colletotrichum cereale*; 12, *Bacterium* spp.

INTERNAL INFECTION

The evidence of internal infection in seed corn showing no external symptoms is suggestive with germinating kernels where the growth of the parasitic fungi is consistently observed at the germinal end. Even this evidence allows for error because of the possibility of surface organisms being present. In resorting to methods of thorough disinfection one still finds the presence of fungous growth appearing on the germinating seeds. The manner in which infection is carried in the seed has not heretofore been extensively investigated. Symptoms of kernels, as we have pointed out for the four parasites described, are easily recognized; however, it is with normal-appearing kernels that we wish to establish the manner of internal infection.

A brief discussion of the morphology of corn may help in explaining the exact nature of this internal parasitism. The kernel, seed, or corn to which it is commonly referred is a ripened fruit or caryopsis. It consists of a mature ovary with all its adnate parts. The kernel is established in a "fruit cup" formed by six fruit envelopes. According to Winton (*39*) this cup consists of—

three glumes—a flowering glume, a palet, and another palet belonging originally to a rudimentary blossom.

The cap of the kernel consists of a dried tissue, nonfunctioning at maturity. The peripheral cells of the cap are continuous with those of the pericarp. They are approximate to the integuments on line with the base of the scutellum. The integuments curve in towards the dorsal end of the scutellum. The inner structure of the cap consists of thick-walled cells and several vascular strands. This cap if partly cut or broken off exposes a small cavity over the dorsal extremity of the scutellum. There is also observed a brown covering adhering to the lower dorsal end of the scutellum. The cavity varies in size, and the covering over the scutellum is light brown to black, according to variety.

The vascular bundles in the cob above the butt have a peripheral distribution, so that the center or pith is made up of only parenchyma cells. The attachment of a pair of ovaries converge to a common base in relation to the vascular system. The ovaries are in direct relation to several bundles which gradually converge into several strands near the base of each ovary. This relation in a young cob can be seen with a hand lens. It is mentioned briefly here since it explains the path of infection if it occurs through the vascular system. If such infection resulted it would seem that the pairs of kernels would be equally infected.

The four fungi mentioned have been found commonly established within kernels which show no symptoms of disease. It was further impossible in many cases to correlate any of the various symptoms of the butts of ears with such infection. The mycelium is found extensively developed in this cavity. In *Fusarium moniliforme* and *Cephalosporium sacchari* abundant internal spore production is found (Pl, 7, A). This can easily be determined with a hand lens or with the low-power objective of the microscope. In some instances where the fungus has progressed to the tissue of the embryo in kernels showing no external symptoms, a free-hand longitudinal section will show a discoloration of the embryo as well as fungus development in the cavity around the radicle and the plumule (Pl. 8 A, B). The development of the fungus from the cap into the germ and endosperm appears to be restricted by the black layer. However, with favorable conditions upon the germinator the fungus becomes active and the seedling is attacked. The feeders, scutellum, and epicotyl first show symptoms of infection.

This internal method of infection was first observed by the junior writer in the spring of 1920. It was observed that when the cap was removed increased germination free from severe infection was obtained. This point was thought to be the result of elimiminating the greater part of the internal fungus infection. It was further established that by disinfecting kernels after removal of cap perfect germination was secured in many instances. Where infection of *Fusarium moniliforme* and *Cephalosporium sacchari* occur the internal development in the cavity under the cap can be recognized with a hand lens. In *F. moniliforme* and *C. sacchari* the abundant internal development of spores is easily determined by the method of crushing the lower germinal end of the kernel and making

a poured agar plate. The numerous colonies appearing indicate the abundant internal spore production.

Many samples of seed corn show slight discoloration of the seed coat near the germinal end. These discolorations become pronounced during germination and assume various colors such as pink, lavendar, or black. The black discolorations were found most consistently with Diplodia infection, the lavendar with *Fusarium moniliforme*, and the pink with *Gibberella saubinetii*. This condition we consider the result of the fungus progressing beyond the cavity and becoming established in the tissue comprising the pericarp. We have observed this condition in kernels before and during germination. This method of internal infection explains the failure to secure disease-free seed by methods of disinfection. The tissue of the cap, even with extensive presoaking, fails to soften materially. The long exposures to various disinfectants are not entirely effective even subsequent to presoaking. Such treatments usually injure the embryo and retard germination.

Cross and longitudinal sections through the germ end of those internally infected kernels show the mycelium also established in the tissue of the cap. In some cases the mycelium is observed among the thin-walled cells near the periphery of the cap. The mycelium is not abundantly established, but it is observed ramifying between as well as penetrating the cells. The thick-walled cells toward the vascular strands do not appear to be penetrated. In several instances long intercellular hyphae were observed in the vascular tissues. The development of the fungus in the cavity is probably subsequent to the infection of the tissue comprising the cap. So far in our studies there was no conclusive evidence to show that such internal infection was always by means of the vascular system as the result of stalk or shank infection.

GENERAL DISCUSSION

With the nature of the internal infection determined, the means by which the fungus gains entrance and its relation to methods of control remain to be established.

According to the occurrence of seed corn infection, we must reckon with two means of infection quite independent of each other.

First: Direct kernel or blossom infection. Silk as the path of infection has been considered a common method for *Diplodia zeae* by Heald, Wilcox, and Pool (*16*) and Van Der Bijl (*38*) and for *Fusarium moniliforme* by Valleau (*36*). No conclusive proof has been advanced to show that infection occurs by means of the stigma. It is considered, in view of the work so far reported, that such infection occurs because the silk stage offers an accessible means of entrance. The silk, becoming dry and shriveling when the corn is in the late milk or dough stage, affords an opportunity for infection by wind-borne spores. Such type of infection apparently accounts for the ears shown in Plate 13. A parallel type of infection appears to be the most common for wheat scab as reported by Adams (*1*). Moisture conditions in the cup before maturity of the ear are no doubt favorable for development of the fungi once they are established from external sources. This tissue of the cap offers little resistance to these parasites and would allow for their establishment and development into the cavity under the cap. The prevalence of infection under these conditions would depend upon exposures of ears in the field to favorable conditions for infection. The study of infected kernels failed to indicate whether infection resulted by this means or from vas-

cular infection through the butt. Even after maturity if the corn is exposed to excessive or constant moisture, the various parasites as well as saprophytes may be established in the storage tissue of the fruit. This we believe in part explains the presence of *Aspergillusniger* and *Penicillium* spp. that frequently appear even after thorough disinfection. They have become established because of unfavorable conditions under which the corn has been exposed and stored. These types of fungi on the germinator may also retard or in some cases inhibit germination. They attack the storage tissue of the scutellum in a way similar to *Rhizopus nigricans*, as described by Adams and Russell (*2*). It is noted that where extensive rotting of kernels occurs on ears it is usually confined to the upper end. The exception in the case of the *F. moniliforme* kernel-rot is difficult to explain. The irregular rotted kernels, as shown in Plate 12, E, may have been individual grains delayed in ripening or ones for some unknown reason susceptible to infection. It is still an open question as to the possibility of direct silk infection.

Second: Indirect seed infection by means of systemic, stalk or shank infection. Smith and Hedges (*31*) have advanced some evidence of systemic infection for *Diplodia zeae*. If such type of infection commonly occurs with these parasites it would seem reasonable to expect poorer stands of corn than are reported. There is no doubt that a systemic infection occurs, but how commonly remains to be determined. Such infection does not necessarily have to follow from the planting of infected seed but may also occur because of soil infestation. Regardless of root infection, the stalk or shank may become points of initial infection. The fungus may in this way follow the vascular tissue into the fruit. This may be the most common means of infection and may explain the internal infection in seeds showing no external symptoms. Hoffer and Holbert (*18*) state that—

> after the first killing frost the stalks and especially the shanks and the ears are invaded rapidly by these disease organisms. Warm weather following the first killing frost favors those ear infections, which greatly reduce the vitality of the seed.

A discolored or "stringy" condition of the butt is very suggestive of vascular infection. An extensive survey has failed to consistently correlate a discolored butt with infection of kernels. A discoloration of the butt may occur subsequent to harvesting as the result of poor storage conditions and many types of black molds found established. When the ear is broken in half and a discoloration of the vascular system is observed, it is very good evidence of an infected ear. The growing point consisting of the preformed nodes may be some distance beyond the foci of infection. After mature growth in height is reached the infection may spread so as to become systemic and penetrate the ears. Inoculations under field conditions should easily establish this point.

The pathogenicity of the four parasites discussed has been definitely established under greenhouse conditions. Over 1,500 plants have been grown to determine seedling infection and for inoculation work. It is planned to duplicate much of the inoculation work under field conditions. Nodal and internodal inoculations have indicated the rapid internal development of these parasites. The importance of each of these parasites requires much more study along lines of inoculation and the activity of the fungus.

Severe internal infection of plants even in the tassel stage has not caused any pronounced external symptoms. The corn plant, because

of its morphological structure, is very capable of offsetting root infection without conspicuous external symptoms.

It would hardly seem practical to resort to any means of removing the cap and then disinfecting for control of these rots of corn. Careful selection in the field and proper care in handling and storage along with germination tests should adequately eliminate the possibility of seed corn infection. The question of soil conditions, fertilization, rotation, and cultural methods should be given particular attention in order to reduce infection and secure strong initial growth.

With further inoculation work duplicated under field conditions it is hoped to prove the points which have been discussed theoretically. In pointing out these possibilities it is hoped that other investigators will assist to determine such proof under their conditions.

SUMMARY

(1) Through an extensive survey of field seed corn in Delaware by means of cultural and germination studies at least four prevalent fungous parasites were determined. A brief survey of field corn from 21 other States indicates a similar prevalence of these organisms.

(2) The following four parasites were consistently found in our studies: *Cephalosporium sacchari* Butler; *Fusarium moniliforme* Sheldon; *Gibberella saubinetii* (Mont.) Sacc. and *Diplodia zeae* (Schw.) Lev. The parasitism of these fungi has been determined by inoculations.

(3) So far as we have been able to determine the fungus here referred to as *Cephalosporium sacchari* is reported for the first time as a parasite of corn. *Fusarium moniliforme* is considered identical with *Oospora verticilloides* described on corn in Italy by Saccardo.

(4) Cultural studies of field corn in Delaware show the following prevalence for these parasites: *Cephalosporium sacchari*, 39.54 per cent; *Fusarium moniliforme*, 19.92 per cent; *Gibberella saubinetii*, 5.95 per cent; *Diplodia zeae*, 5.69 per cent.

(5) Inhibition of germination as the result of virulence by these parasites occurs in the following order under cultural and laboratory conditions, *Diplodia zeae*, *Gibberella saubinetii*, *Fusarium moniliforme*, and *Cephalosporium sacchari*.

(6) Species of the following genera of fungi have also been found established internal of corn: Aspergillus, Cladosporium, Penicillium, Alternaria, Helminthosporium, Rhizopus, Spicaria, Hormodendrum, Torula, Chaetomium, Colletotrichum, and also several bacteria. These fungi are not considered in the same degree of importance as the above-mentioned parasites. It is probable they have become established as the result of unfavorable field and storage conditions.

(7) The method by which these parasites are carried in the kernel was determined through cultural and histological studies. It was found that the parasites become established in the tissue of the cap and the cavity between the cap and dorsal point of the scutellum. In some instances the fungus works upward underneath the pericarp. Under certain favorable conditions not yet determined the fungus infects and destroys the embryo.

(8) Seed disinfection has not proved successful because of the manner of internal infection.

(9) No uniform symptoms to associate infection of field corn with these parasites can be consistently described. Various discolorations of the germ end of the kernel are found. These symptoms are always

associated with infected seed. Normal-appearing kernels as well as ears may be infected without any external symptoms.

(10) No conclusive evidence was secured which indicates the manner in which infection becomes established in the seed.

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PLATE 1

A.—*Cephalosporium sacchari*, showing typical conidia from host material. The spores are shortly oval to ovoid.

B.—*Cephalosporium sacchari*, showing conidia prior to germination and during early development of germ tube. The conidia commonly become septate prior to germination.

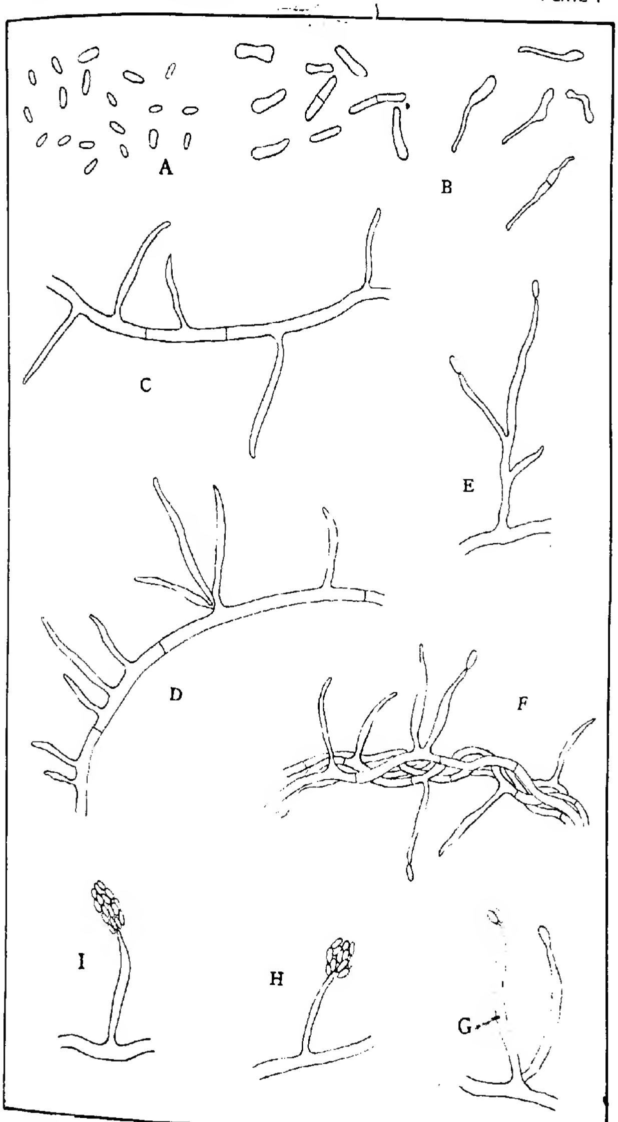
C, D.—*Cephalosporium sacchari*, illustrating the origin of the conidiophores. These are often scattered along the mycelium or a number become grouped as in D.

E.—*Cephalosporium sacchari*, showing a branched conidiophore from growth in culture.

F.—*Cephalosporium sacchari*, showing an intertwined mass of hyphae from a culture. These strands often appear as a definite coremial like growth.

G–I.—*Cephalosporium sacchari*, showing stages in the development of the glomerules. These heads under low power appear similar to those developed by *Fusarium moniliforme*. The spores are not held together in a slime.

Drawn with the aid of the camera lucida, approximately × 600.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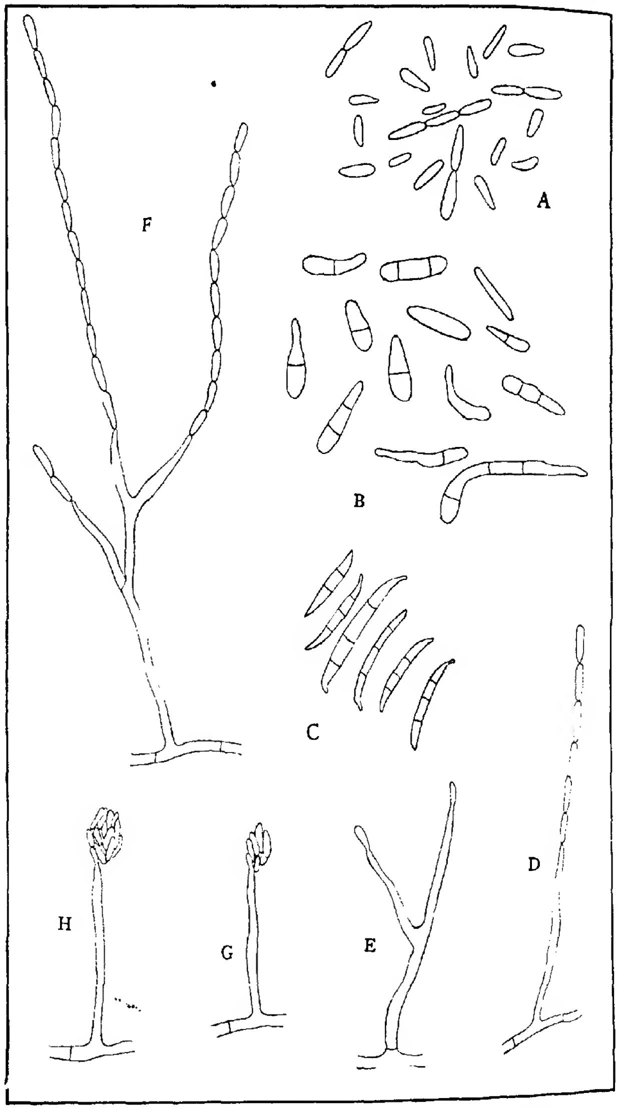
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PLATE 2

A.—*Fusarium moniliforme*, showing typical mircroconidia from host material. They are slightly pyriform and the attenuated end of the spore is often sharply muticate.

B.—*Fusarium moniliforme*, showing mircroconidia during early stages of germination. The spores enlarge considerably and often become septate before the germ tube has developed.

C.—*Fusarium moniliforme*, showing typical macroconidia. These were observed to be mostly three septate. They are sparingly developed and were observed in cultures on rice agar and steamed corn meal.

D, E.—*Fusarium moniliforme*, showing simple and branched conidiophores.

F.—*Fusarium moniliforme*, showing a branched conidiophore with typical catenulate microconidia.

G, H.—*Fusarium moniliforme*, showing two simple conidiophores with the microconidia in heads. These heads are commonly found in old cultures but the spores are not held together in a slime.

Drawn with the aid of the cameralucida, approximately ×600.

26550—23——3

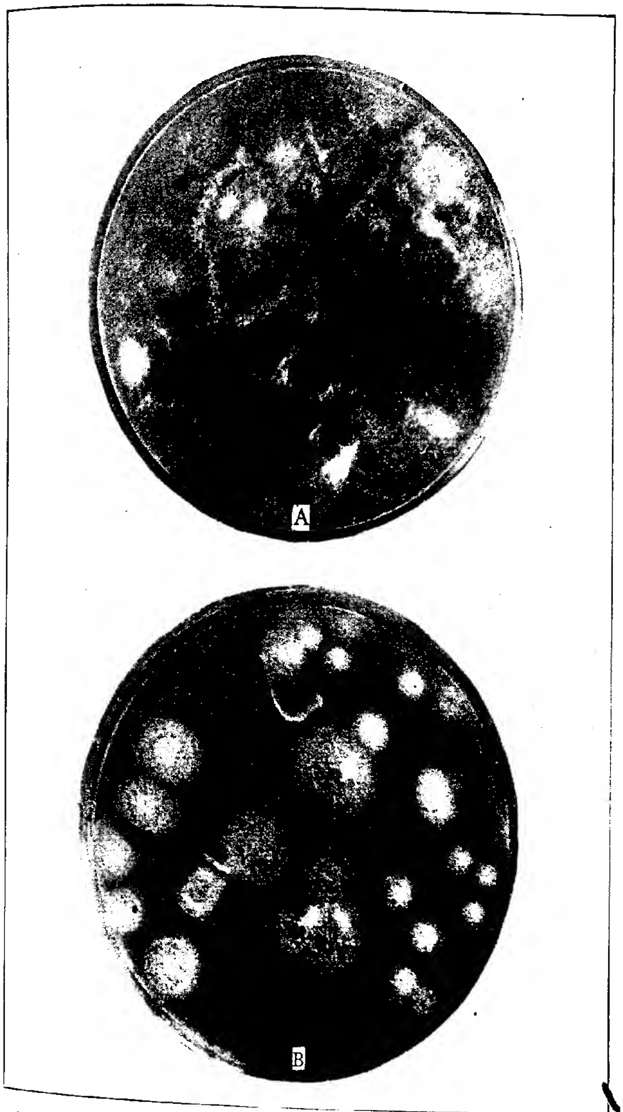
PLATE 3

A.—Culture of *Fusarium moniliforme* in nutrient dextrose agar 3 weeks old, direct from surface-sterilized kernel from which point was cut off and crushed. The remaining part of the kernel was also left in the culture dish. In submedium a deep lavender is formed.

B.—Culture of *Cephalosporium sacchari* in nutrient dectrose agar, 3 weeks old, direct from surface, sterilized kernel from which point was cut off and crushed. The remaining part of the kernel was also left in the culture dish. The colonies on this medium take on a salmon color.

Somewhat reduced.

PLATE 3



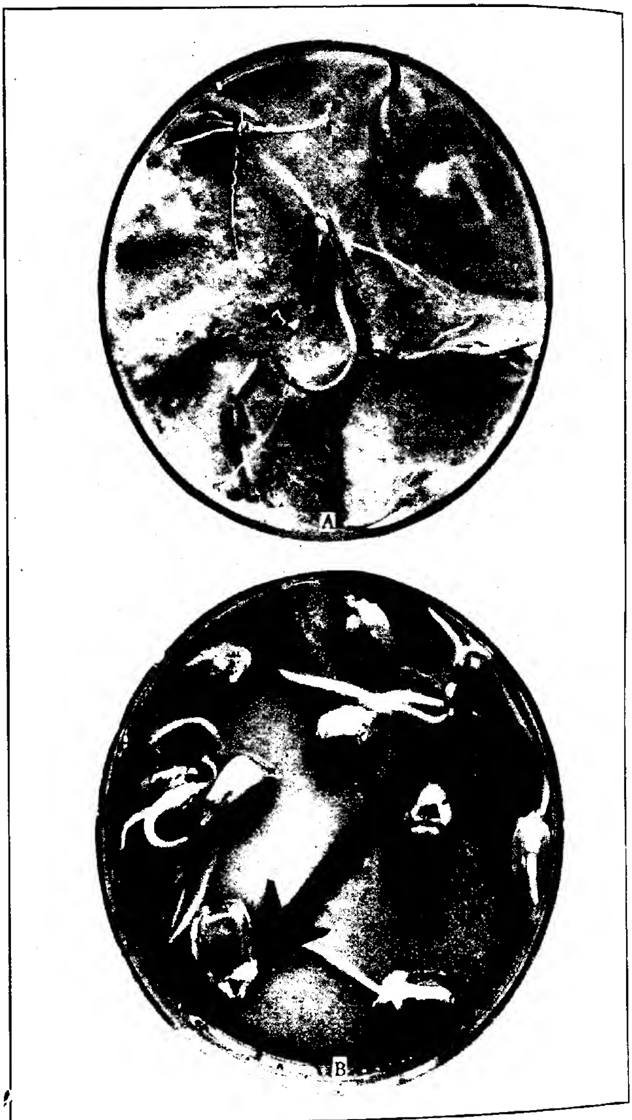


PLATE 4

A.—Cultures of *Diplodia zeae* coming from surface-sterilized corn germinating in culture dish on 20 cc. of nutrient dextrose agar. Ten kernels are taken from each sample and germinated this way. The effect of the parasite is here shown: 40 per cent of the kernels are dead, 50 per cent are germinating weak, and only 10 per cent are strong.

B.—Cultures of *Cephalosporium sacchari* coming from surface-sterilized corn on nutrient dextrose agar. The arrow points to a culture showing the typical bristle or coremial growth. Some of the kernels are so severely infected that slight retardation of growth is noticeable.

Somewhat reduced.

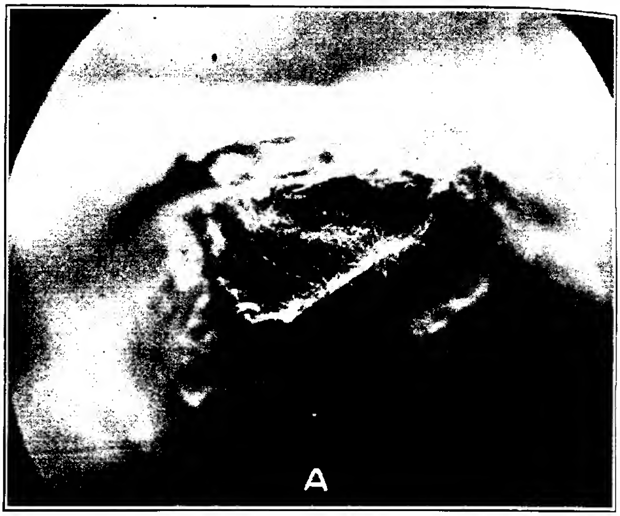
PLATE 5

A.—Sample of corn free from internal paratic fungi germinating on nutrient dextrose agar. The kernels were surface-disinfected with 0.1 per cent bichlorid of mercury in 50 per cent alcohol for one minute, then washed twice in sterile water and immediately placed on the sterile medium.

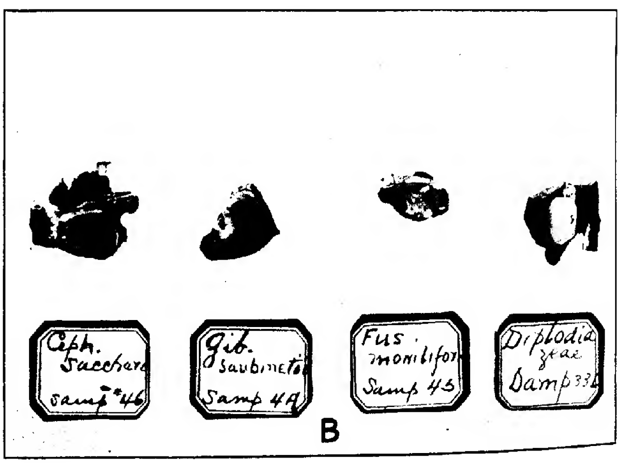
B.—Sample of corn carrying internally two fungi. The black growth is an *Aspergillus* sp. which is present to the extent of 100 per cent; the white fungus, indicated by the arrow, is *Fusarium moniliforme* which is present to the extent of 40 per cent. The presence of the *Aspergillus* sp. probably indicates that the corn had poor curing conditions.

Somewhat reduced.


A
B



A


Ceph.
Sacchar
Samp #46
Gib.
saubineti
Samp 4A
Fus.
monilifor
Samp 45
Diplodia
zeae
Samp 33

B

PLATE 6

A.—Photomicrograph of tip end of kernel with cap removed, showing the presence of *Diplodea zeae* in the cavity, from sample 33D. Each of the several parasitic fungi here described harbor internal of the kernel in this manner. ×30.

B.—Photograph of mounted kernels with caps removed, ready for taking photomicrographs as illustrated above. Somewhat reduced.

PLATE 7

A.—Photomicrograph of the tip end of kernel with cap removed, showing the presence of *Cephalosporium sacchari* in the cavity. Both *C. sacchari* and *Fusarium moniliforme* fruit (microconidia) are borne abundantly in the cavities under the cap. The thickness of the cap prevents disinfectants from destroying the internal fungus. From sample No. 46. ×30.

B.—Photomicrograph of the tip end of a kernel with cap removed, showing the presence of *Gibberella saubinetii*, from sample 4 A. ×30.


A


A

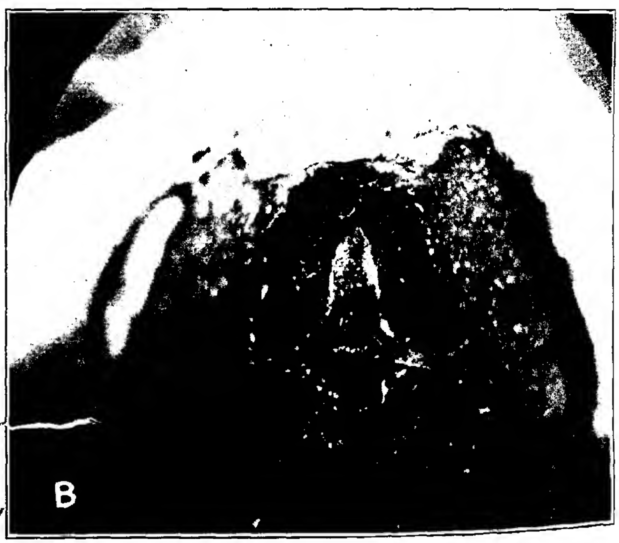

B

PLATE 8

A.—Kernel from sample 321, infected with *Gibberela saubinetii*, in which the parasite has reached the germ and the cavities about the germ. Whenever the germ is invaded by any of the several parasites herein described, germination is either inhibited or greatly retarded. Eighty per cent of the kernels in this sample would not germinate. ×30.

B.—Kernel from Ear 10 B, infected with *Diplodia zeae*, in which the parasite has reached the germ and the cavities about the germ. (See Pl. 9, A.) Seventy per cent of the kernels on this ear were killed by this type of infection; the ear on the surface appeared normal. ×30.

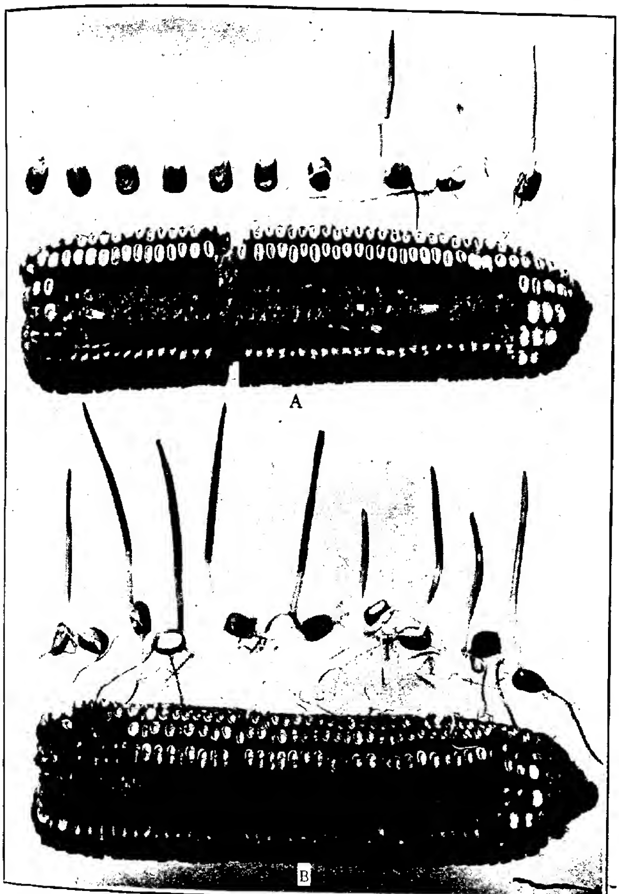
PLATE 9

Two ears of seed corn from the same source. Both ears appeared perfectly normal externally but germination in the modified rag doll showed great difference.

A.—Actual germination of ear 10 B in rag doll; 70 per cent were dead. Culture studies (see Pl. 10) showed 70 per cent of the kernels internally infected with *Diplodia zeae*, 20 per cent with *Gibberella saubinetii*, and 80 per cent with *Cephalosporium sacchari*. *Diplodia zeae* was the active agent in reducing germination.

B.—Actual germination (100 per cent) of ear 10 A, from same source as ear 10 B above. Culture studies showed 100 per cent infection with *Cephalosporium sacchari* and 40 per cent infection with *Gibberella saubinetii*.

Somewhat reduced.



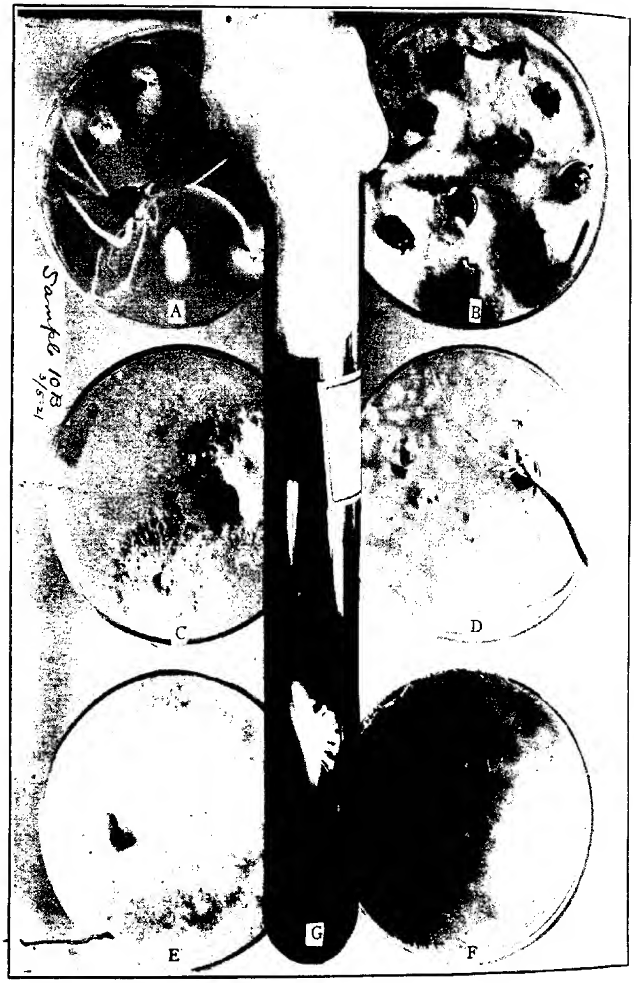

A
B
C
D
E
F
G

PLATE 10

Results of the culture method of determining internal fungi in seed corn. Fifteen kernels in each sample were surface-disinfected and washed in sterile water; 10 of these kernels were placed in one dish on 20 cc. of nutrient dextrose agar. Five kernels were placed each in a separate sterile dish, and the points of each of these were cut off, crushed, and plated. The fungi which were internal readily developed. The culture work on sample 10 B is here shown, in which 70 per cent of the kernels were infected with *Viplodia zeae* (See cultures in dishes marked B, C, D, and E); 20 per cent of the kernels were infected with *Gibberella saubinetii* (See dish marked F.); 80 per cent of the kernels were infected with *Cephalosporium sacchari* (See dishes marked A, D, and E). In this sample *Diplodia zeae* was the fungus most active in inhibiting germiniation. G, coremial growth produced by *Cephalosporium sacchari*. The coremial growths are not usually so pronounced as this.

Somewhat reduced.

PLATE 11

A.—Seedlings germinated in modified rag doll from kernels of sample 181-c.

B.—Sample 181-c. This sample carried internal infection with *Fusarium moniliforme* to the extent of 100 per cent and *Cephalosporium sacchari* 100 per cent and yet gave 100 per cent germination and very strong seedlings in the rag doll. Injury and retardation of germination take place only when the parasitic fungi come in contact with the germ of germinating seedlings.

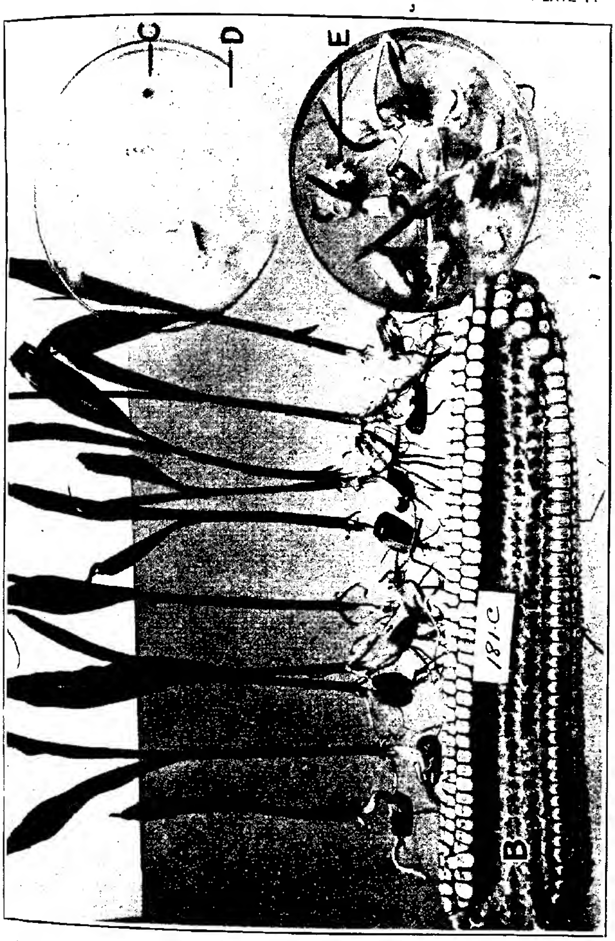
C.—Culture of *F. moniliforme*, internal of same kernel as D.

D.—Culture of *C. sacchari*, internal of same kernel as C.

E.—Ten surface-sterilized kernels placed on 20 cc. of nutrient dextrose agar, all showing both *F. moniliforme* and *C. sacchari*. Only one kernel failed to germinate in the culture dish; 100 per cent gave strong germination in the modified rag-doll germinator.

Somewhat reduced.

PLATE II



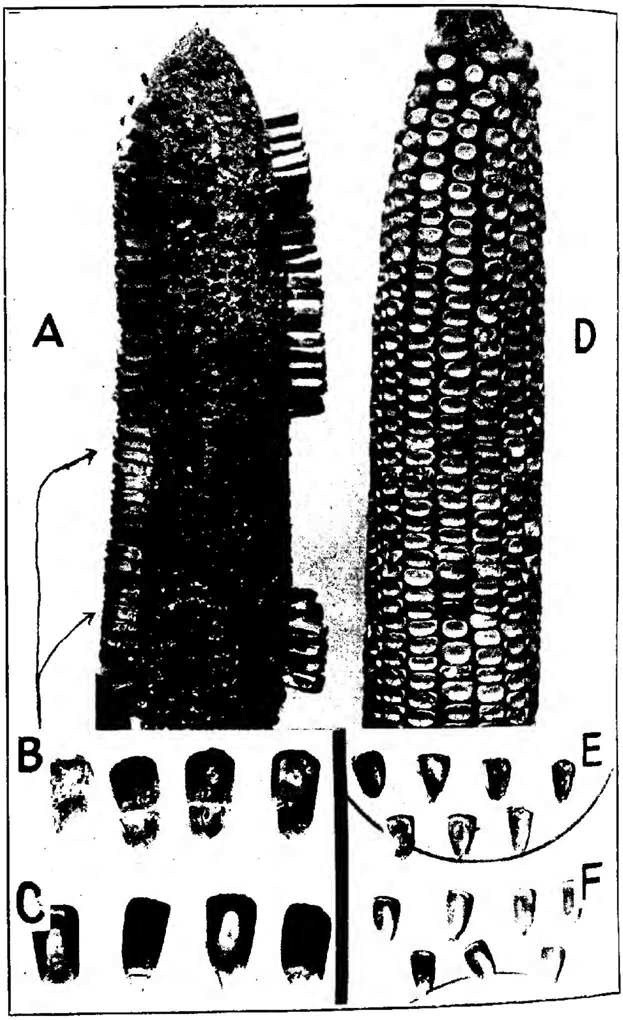

A
D
B
E
C
F

PLATE 12

A.—Ear affected with kernel crack at areas indicated by the arrow.

B.—Injured kernels from ear shown in A.

C.—Uninjured kernels from ear shown in A.

More than 60 per cent of the kernels on the same ear were not injured, as indicated in C. Some of the cracked kernels will germinate. Associated with this cracking are often found *Fusarium moniliforme*, *Cephalosporium sacchari*, and occasionally a *Spicaria* sp. The cause of the cracking is yet to be determined.

D.—Ear affected with a typical kernel-rot caused by *F. moniliforme*.

E.—Rotted kernels from ears shown in D. Such rotted kernels will not germinate. Associated with such rotting is often the lavender discolorations characteristic of the growth of *F. moniliforme*.

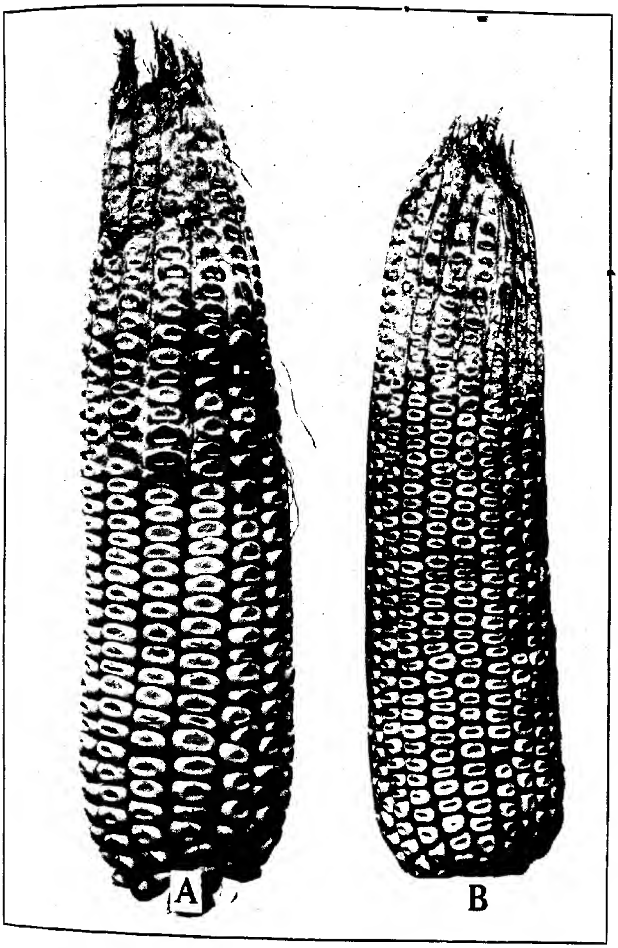
F.—Healthy kernels from ear shown in D.

Somewhat reduced.

PLATE 13

Typical ear rot resulting from natural infection with *Gibberella saubinetii*. In this type of rot it seems probable that infection takes place through the silk mass in the late milk or dough stage. Cultures of the kernels near the base or butt of ear A, gave no evidence of *Gibberella saubinetii* internal, possibly indicating that infection was from above.

Somewhat reduced.


A
B

COTTON ROOTROT IN ARIZONA[1]

By C. J. King

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It has been generally accepted that the fungus, *Ozonium omnivorum*, described by Pammel[2] in 1889, is the cause of the disease known as Texas rootrot, but definite proof has been lacking. The records available show that but little has been added to the knowledge of the life history of the fungus since it was first studied by Pammel,[2] a notable exception being the work of Duggar[3] in 1916, who described a conidial stage from material which he found in a cotton field near Paris, Tex., in 1915. However, Duggar's classification, based on the character of this fruiting form, does not appear to have been adopted by other pathologists.

The conditions at Sacaton, Ariz., during the seasons of 1921 and 1922 permitted unusually satisfactory observations upon the intimate relationship existing between the disease and a fruiting form of the fungus, which is evidently identical with that described by Duggar. In the Salt River Valley, Ariz., during the seasons 1917 to 1919 the fruiting fungus had been observed on several occasions, sometimes in abundance following a rainy period and often as isolated spots where a depression or crack in the soil afforded favorable conditions; but at no time was the material so abundant and so definitely associated with the presence of the disease as at Sacaton during the past two seasons.

In comparing the manner of spread of the disease in various crops, it was noted that in alfalfa fields its behavior was very suggestive of fairy rings. The perfectly formed circles, consisting of an outer ring of recently dead plants, an inner ring, or "bare zone," where only the old stubble of dead plants remains, and a central zone occupied by reestablished plants arising from fragments of crowns or roots not fully destroyed, establish the fact that the disease spreads from a center in ever-widening circles and that having passed a given place leaves this spot free from the disease until reinfected. In badly infected fields where the disease is of long standing the crossing and recrossing of the widening rings may completely obscure the fairy-ring effect.

The resemblance to fairy rings is made still more striking by the abundant crops of fruiting bodies, which in Arizona appear on the periphery of the circles shortly after the occurrence of rainy weather. The newly formed fruiting bodies appear as feltlike mats on the surface of the ground, or in cracks or depressions, and are rarely found more than 6 or 8 inches from the outer circle of recently wilted plants. At times they have been so abundant as to cover over 300 square feet of soil surface in a 22-acre alfalfa field where 3 or 4 acres of the plants had died.

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[1] Accepted for publication Dec. 16, 1922.

[2] Pammel, L. H. COTTON ROOT-ROT. *In* Tex. Agr. Exp. Sta. 2d Ann. Rpt. 1889, p. 61–86, 3 pl. 1890.

[3] Duggar, B. M. THE TEXAS ROOT-ROT FUNGUS AND ITS CONIDIAL STAGE. *In* Mo. Bot. Gard. Bul., v. 3, p. 11–23, 5 fig. 1916. Bibliography, p. 23.

Journal of Agricultural Research,
Washington, D. C.
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Vol. XXIII, No. 7
Feb. 17, 1923
Key No. G–281

CONTROL EXPERIMENTS

The characteristic behavior, indicating that the disease is virulent only near the rim of the circles, encouraged the writer's attempted use of certain fungicides in the effort to check the spread of the fungus. By thoroughly saturating the soil to a depth of 4 feet with a solution of formaldehyde (1 part 40 per cent formalin to 100 parts water), in small circular areas where the activity had just started, it was found possible to prevent further spread of the disease. It appears that the fungus mycelium must extend a foot or more in advance of the last plant showing distress, since to be effective the soil 18 inches outside the apparent periphery in alfalfa fields must also be included in the treatment, it was found.

With cotton plants the mycelium appears to extend for at least 2½ feet in advance of the ring of recently wilted ones, there being instances where the disease reappeared after treatment on plants 2½ feet away. In no case in cotton fields has the disease reappeared where the treated area extended as far as 3 feet outside the ring of recently wilted plants. The most practical method of applying the solution seems to be that of throwing up a small dike around the area to be treated and pouring in the solution gradually until the soil has been saturated to a depth of at least 4 feet.

None of the areas in alfalfa fields treated as described above in July and early August, 1922, have shown further disease activity after 60 days. With the control areas where dikes were thrown up, but no fungicide applied, the disease has progressed from 4 to 4½ feet during that time. There has been no reappearance of the disease outside any of the areas in cotton fields during 50 days after treatment. In the control areas the disease has advanced from 6 to 8 feet during that interval.

The first successful attempt to communicate the rootrot disease to healthy cotton plants was made in August, 1922. The inoculation experiments were conducted with normal plants in the field, since it had been found difficult to grow normal cotton plants or develop them into a fruiting condition in pots in the greenhouse under the climatic conditions which prevail at Sacaton in summer. An area was selected on which there had been no evidence of rootrot since the experiment station was established in 1907. A trench was made on July 29, approximately 2 feet wide, 9 feet long, and 2½ feet deep, between two cotton rows in which the plants were normal in every respect and fruiting heavily. With a small hand pick the soil on one side of the trench was dug away from the roots of each plant alongside the trench until 15 inches or more of each tap root had been exposed in the small vertical channels. On this side of the trench sections of rootrot-diseased cotton roots 1 inch in length, previously washed in distilled water, were inserted in the channels and placed end to end and in direct contact with the healthy tap root. The full length of the channel was then stuffed with sterile cotton soaked in distilled water, and then soil, which had been saturated with water, was applied with a trowel on top of the cotton until the channels were completely filled.

On the other side of the trench the roots were exposed in the same manner, but instead of diseased roots being used the cotton wrapping was soaked in distilled water containing 50 cc. per liter of fresh spores collected from active rootrot spots. After 21 days following a rainy period, one of the plants treated with diseased roots wilted suddenly and upon

being pulled up showed in abundance the characteristic mycelium of the rootrot fungus. Within 5 days four other inoculated plants died in the same characteristic manner, but by this time the soil on the sides of the trench had become so depleted of moisture that the disease was unable to make further progress.

From the first inoculated plant which died (it was pulled up immediately after wilting), the mycelium was isolated and pure cultures were prepared after the method of Atkinson.[4] Bits of sterile cotton roots were used as the medium. After abundant growth had been obtained from one of these pure cultures on a layer of sterile cotton roots in moist chambers, the material was conveyed to another trench that had been prepared a few feet from the first. This material was then used to inoculate 10 plants on one side of the trench, 4 plants being left as a control. The 14 plants on the other side of the trench were treated with diseased roots as described above. The plants were kept well supplied with moisture and in 12 days they began dying almost simultaneously on both sides of the trench. By September 20, the disease had appeared in all but 4 or 5 of the plants on both sides of the trench, including the four control plants, which had been attacked one at a time by the invasion of the mycelium through the soil from the inoculated plants. By October 1, the mycelium had spread from the pure-culture treatment to the plants 3½ feet away in the next row, and 4 of them had succumbed by this date. After seven weeks none of the plants which had been treated with spores showed any signs of distress.

Spores collected from fresh fruiting mats were induced to germinate in distilled water and in artificial media after several days, but the resulting growth was extremely slow. After five weeks in the Van Tieghem cells the germ tubes have grown but two or three times longer than the diameter of the spores. The difficulty in inducing the spores to develop in artificial media and the failure to induce the disease by applying them to the roots of healthy plants is not surprising. If the conditions necessary for the development of spores were not very exacting, the enormous quantities in which they are produced would long since have made the distribution of the disease universal.

It is of the utmost importance to determine as promptly as possible to what extent the promising results obtained in these local experiments are applicable in other regions.

SUMMARY

(1) At Sacaton, the fungus causing rootrot of cotton and alfalfa fruits abundantly in favorable seasons, the fruiting masses being identical apparently with those described by Duggar.[5]

(2) The fungus spreads from a center in ever-widening circles after the manner of a fairy-ring fungus.

(3) Plants have been inoculated successfully with pure cultures of the mycelium.

(4) Spores are germinated with difficulty and have not been used successfully as a medium of inoculation.

(5) The spread of the disease may be checked by the use of formaldehyde.

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[4] ATKINSON, G. F. METHOD FOR OBTAINING PURE CULTURES OF PAMMEL'S FUNGUS OF TEXAS ROOT ROT OF COTTON. *In* Bot. Gaz., v. 18, p. 16–19. 1893.

[5] DUGGAR, B. M. OP. CIT.

CORRELATIONS BETWEEN VARIOUS CHARACTERS OF WHEAT AND FLOUR AS DETERMINED FROM PUBLISHED DATA FROM CHEMICAL, MILLING, AND BAKING TESTS OF A NUMBER OF AMERICAN WHEATS [1]

By Jacob Zinn [2]

Formerly [3] Associate Biologist, Maine Agricultural Experiment Station

INTRODUCTION

From a plant breeder's point of view it would be very desirable to establish an index by means of which the baking quality or strength of wheat flour could be determined at an early stage of the breeding work so that the weak wheat strains could then be discarded, instead of carrying them through all the stages of propagation and the processes in the bakehouse. The solution of this problem calls for the establishment of a definite relation between a wheat character, determinable early in the selection or breeding work, and the strength of the flour as measured by the loaf volume.

A study of the literature on the chemistry of wheat (*18*) [4] reveals a marked divergence of views as to the significance of the various chemical characteristics of wheat in relation to the bread-making quality of the flour. Almost without exception, the work on the relationship of the chemical characters to bread-making value is characterized by the lack of statistical methods. The writer is aware of no publication in which this relationship and its degree are expressed in terms which will allow of comparison.

In the present paper a large number of published data on the chemistry of wheat are compiled and the coefficients of correlation for the important chemical characters computed. In submitting the results of this study attention must be called to the limitations of the material. In order to exclude possible errors due to differences in the nature of the material and in technic, it was thought advisable to analyze the data into groups by States and according to the kind of wheat rather than to lump them together into larger classes including several States or wheat districts. This, of course, resulted in a reduction of the data in the individual groups, which in some correlation tables reaches a point where conclusions can not always safely be drawn.

A further limitation of the material considered in this study lies in the relatively small amount of data on the chemistry of wheats and flours derived from pure strains. The data on pure lines are limited both in regard to localities and number of samples analyzed. The writer believes that the proper approach to the solution of the question of correlations

[1] Accepted for publication Nov. 14, 1921.
Wheat Investigations II. Papers from the Biological Laboratory of the Maine Agricultural Experiment Station, No 147.

[2] In the preparation of this paper the writer received helpful suggestions from Dr. John W. Gowen, of the Maine Agricultural Experiment Station, and assistance from Mr. H. C. McPhee and Mr. E. R. Ring, also of the Maine station, in the tabulation and computation of the data.

[3] Dr. Jacob Zinn died in October, 1921.

[4] Reference is made by number (italic) to "Literature cited," p. 548.

Journal of Agricultural Research,
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Vol. XXIII, No. 7
Feb. 17, 1923
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between the characters here considered must be based upon a large amount of data secured from analyses of pure lines and made by the same laboratory, rather than any analyses of commercial varieties. In the light of this proposition the present paper is to be regarded as a preliminary study, as the amount of data from pure lines here included is very limited.

Notwithstanding these limitations the present study is of some interest. It represents the first attempt to analyze statistically a large amount of raw data by computing from them correlation coefficients for a number of characters. It should throw some light upon the relationships of the chemical characters where formerly they were based merely upon observation or inspection of data. It should also be instructive to compare, in the future, the results obtained in the main from commercial varieties with those to be secured from pure lines of wheat. Finally, it may furnish suggestions of service in appraising the bread-making value of wheats.

MATERIAL AND METHODS

The material used in the present study is the data from chemical, milling, and baking tests published by the agricultural experiment stations of Colorado (*7*), Idaho (*8*), Kansas (*16*), Maine (*18*), Minnesota (*2, 3, 4, 5*), Montana (*13*), North Dakota (*6, 9, 10, 15*), Ohio (*1, 17*), Wisconsin (*11*), and the Central Experimental Farms of Canada at Ontario (*12*). While the choice of the material was naturally limited by the availability or lack of data for the different grain-growing regions, an inspection of the localities stated above will show that they include the important districts of the hard red spring and semihard red spring wheats, the hard red winter, and semihard red winter wheats. No data are included on the white soft wheats of the Pacific coast, the soft winter wheats of the Mississippi district, and the durum wheats.

The classification of the material is first based upon the geographic distribution of the wheats and flours, the data of each experiment station being treated separately. The data of each experiment station are further subdivided according to the kind of wheat, the spring wheats and winter wheats being grouped separately. A further subdivision of the material was effected by grouping the spring wheats and winter wheats according to whether they are commercial varieties or pure strains.

The coefficients of correlation computed in this study relate to 10 important values secured from chemical analysis and milling and baking tests. Protein content in wheat is correlated with protein content in the flour, dry gluten, gliadin, and yield of flour. Loaf volume is correlated with protein content in wheat, protein content in the flour, dry gluten content, wet gluten content, quality of gluten, gliadin water absorption, and yield of flour. Dry gluten is correlated with gliadin and water absorption. As already noted, the whole material was analyzed into four groups: Spring wheats, winter wheats, commercial varieties, and pure strains. Of the total number of 100 computed coefficients of correlation, 30 relate to commercial varieties of spring wheat, 27 to pure strains of spring wheat, 32 to commercial varieties of winter wheat, and 11 to pure strains of winter wheat.

Since the data used in this paper have all been published by the experiment stations enumerated above, it is not necessary to publish the large number of correlation tables. However, for each kind of correlation, with a few exceptions, a representative correlation table is here

published, giving the frequency distribution typical for each pair of characters. Following each table are tabulated summaries of correlation coefficients pertaining to the characters correlated in the preceding table. The calculation of the coefficients of correlation was carried to the sixth decimal place and recorded to the fourth.

ANALYSIS OF DATA

PROTEIN CONTENT IN WHEAT AND FLOUR

From the nature of these two values a very close relation between them may be anticipated. The degree of that relationship is of interest.

TABLE I.—*Correlation between protein content in the wheat and the flour of Minnesota spring wheats of commercial varieties*

Protein in the wheat (per cent).	Protein in the flour (per cent).												Total
	9.00 to 9.50	9.50	10.00	10.50	11.00	11.50	12.00	12.50	13.00	13.50	14.00	14.50 to 15.50	
9.5 to 10.00	...	...	...	...	...	...	...	...	...	...	...	...	...
10.00	...	1	...	...	...	...	...	...	...	...	...	...	1
10.50	1	6	2	...	...	...	...	...	...	...	...	...	9
11.00	3	4	6	4	...	...	...	...	...	...	...	...	17
11.50	...	...	10	10	3	2	...	...	...	...	...	...	25
12.00	...	...	...	11	21	6	1	...	...	...	...	...	39
12.50	...	...	2	2	13	17	1	1	...	...	...	...	36
13.00	...	...	...	2	1	7	9	1	1	...	...	...	21
13.50	...	...	...	...	...	3	15	6	4	...	1	...	29
14.00	...	...	...	...	...	1	1	3	7	1	...	...	13
14.50	...	...	...	...	...	...	...	...	3	2	2	1	8
15.00	...	...	...	...	...	...	...	...	...	...	3	1	4
15.50	...	...	...	...	...	...	...	...	...	3	1	1	5
16.00	...	...	...	...	...	...	...	...	...	1	...	...	1
16 to 16.50	...	...	...	...	...	...	...	...	...	...	...	...	...
Total	4	11	20	29	38	36	27	11	15	7	7	3	208

$r = 0.9120 \pm 0.0079$.

TABLE II.—*Correlation coefficients for protein content in the wheat and the flour*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
Colorado spring wheats	48	0.8036±0.0344
Minnesota spring wheats	208	.9120± .0079
North Dakota spring wheats	119	.9161± .0100
Montana spring wheats	34	.9710± .0066
Kansas winter wheats	47	.8957± .0194
Minnesota winter wheats	43	.9216± .0154
Montana winter wheats	91	.9259± .0101
Ohio winter wheats	99	.9438± .0074
Idaho winter wheats	60	.9728± .0046
Pure strains:		
Minnesota spring wheats	48	.8921± .0199
North Dakota spring wheats	28	.9686± .0078
Wisconsin winter wheats	7	.8814± .0571
Ohio winter wheats	25	.9148± .0220

The data in Tables I and II show a very high positive correlation between protein content in wheat and flour. The coefficients of correlation range from 0.8036 ± 0.0344 for the Colorado spring wheats to 0.9728 ± 0.0046 for the Idaho winter wheats. The average for the commercial varieties is almost identical with that for the pure strains.

DRY GLUTEN AND PROTEIN IN THE WHEAT

In view of the significance attached to the gluten content as influencing the strength of flour, the relation of the former to the protein content in the wheat is of interest.

TABLE III.—*Correlation between dry gluten and protein content in Ohio winter wheats of commercial varieties*

Dry gluten content (per cent).	Protein in the wheat (per cent).									Total.
	8.50 to 9.00	9.00	9.50	10.00	10.50	11.00	11.50	12.00	12.50 to 13.00	
6.50 to 7.00										
7.00	4	1								5
7.50	1	2	2							5
8.00		4	2		1	1				8
8.50			2	3	3	1				9
9.00				1	3	3	3			10
9.50						1	4	2		7
10.00						3	3	1		7
10.50							3	1	1	5
11.00										
11.50										
12.00									1	1
12.50								1		1
13.00 to 13.50										
Total	5	7	6	4	7	9	13	5	2	58

$r = 0.8695 \pm 0.0217$.

TABLE IV.—*Correlation coefficients for characters dry gluten and protein content in wheat*

Kind of wheat.	Number of samples.	Coefficients of correlation.
Commercial varieties:		
Colorado spring wheats	48	0.6951 ± 0.0503
North Dakota spring wheats	37	.8300 ± .0345
Ohio winter wheats	58	.8695 ± .0217
Idaho winter wheats	60	.9824 ± .0031
Pure strains:		
Maine spring wheats	31	.9603 ± .0095
Ohio winter wheats	13	.9483 ± 0.0190

The correlation between these two chemical characters is positive and very high, as would be expected. The correlation coefficients range from 0.4951 ± 0.0503 for the Colorado spring wheats to 0.9824 ± 0.0031 for the Idaho winter wheats, the data for the two States marking again the limits of the range as in the case of the relationship of the protein in wheat to protein in the flour.

GLIADIN AND PROTEIN CONTENT IN THE WHEAT

Gliadin, both its absolute amount in the flour and in the form of the gliadin number (ratio of gliadin to glutenin) has by some chemists been regarded as the determining factor of strength of the wheat flour. The degree of its association with the crude protein content of wheat should, therefore, be a matter of interest in the study of the chemical relationships in wheat.

TABLE V.—*Correlation between gliadin and protein content in North Dakota spring wheats of commercial varieties*

Gliadin (per cent).	Protein in wheat (per cent).																				Total.
	9.00 to 9.50	9.50	10.00	10.50	11.00	11.50	12.00	12.50	13.00	13.50	14.00	14.50	15.00	15.50	16.00	16.50	17.00	17.50	18.00	18.50 to 19.00	
3.50 to 4.00																					
4.00													1								1
4.50	1												1								2
5.00					1	1															2
5.50		1	2	1	1	2	1	1		1											10
6.00						1	4	3	2												10
6.50							2	7	3	2		2									16
7.00										5	2	4	3	1		3	1				19
7.50										4	6	2	2	3				1			18
8.00												2	3	2		2				1	10
8.50												4	1	3	2	5			1		16
9.00															1		2	2			5
9.50																1	1		1	1	4
10.00 to 10.50																					
Total	1	1	2	1	2	4	7	11	5	12	8	14	11	9	3	11	4	3	2	2	113

$r=0.7863\pm0.0243$.

TABLE VI.—*Correlation coefficients for gliadin and protein in wheat*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	113	0.7863 ± 0.0243
Kansas winter wheats	37	$.8508\pm.0306$
Ohio winter wheats	40	$.9244\pm.0160$
Pure strains:		
North Dakota spring wheats	26	$.9295\pm.0181$

The data in Tables V and VI show a very close correlation between gliadin and crude protein in wheat. The correlation coefficients range from 0.7863 ± 0.0243 for the North Dakota commercial varieties of spring wheat to 0.9295 ± 0.0181 for the North Dakota pure strains of spring wheat.

The characters so far discussed are all component parts of the crude protein of wheat, and their mutual relationship is to be *a priori* assumed. It is of interest, however, to note the intensity of this relationship as given by the correlation coefficients.

FLOUR YIELD AND PROTEIN IN WHEAT

The question of relation between flour yield and protein in the wheat is of concern especially to the miller.

TABLE VII.—*Correlation between flour yield and protein in Minnesota spring wheats of commercial varieties*

Protein in wheat (per cent).	Yield of flour (per cent).												Total.
	65 to 66	66	67	68	69	70	71	72	73	74	75	76 to 77	
9.50 to 10.00	...	...	...	...	...	...	...	...	...	...	...	...	...
10.00	...	...	...	...	...	...	1	...	...	...	...	...	1
10.50	1	...	...	1	1	3	1	1	1	...	...	...	9
11.00	1	...	1	2	3	6	...	3	1	...	...	...	17
11.50	1	...	3	...	3	5	5	...	7	...	1	...	25
12.00	...	1	1	...	3	6	10	8	6	1	3	...	39
12.50	...	1	...	...	4	7	6	7	3	3	...	2	33
13.00	...	...	1	1	1	3	3	3	6	1	1	...	20
13.50	...	...	1	1	3	2	5	4	11	1	...	...	28
14.00	...	1	1	...	...	3	1	1	5	...	...	...	12
14.50	...	...	...	...	2	2	...	...	3	1	...	...	8
15.00	...	...	...	...	2	1	...	...	...	1	...	...	4
15.50	...	...	...	...	2	...	...	...	3	...	...	...	5
16.00	...	...	...	...	...	...	...	...	1	...	...	...	1
16.50 to 17.00	...	...	...	...	...	...	...	...	...	...	...	...	...
Total	3	3	8	5	24	38	32	27	47	8	5	2	202

$r = 0.1789 \pm 0.0459$.

TABLE VIII.—*Correlation coefficients for protein in the wheat and yield of flour*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	99	-0.2167 ± 0.0646
Minnesota spring wheats	202	$.1789 \pm .0459$
Montana spring wheats	34	$.3751 \pm .0994$
Ohio winter wheats	29	$-.2062 \pm .1199$
Montana winter wheats	91	$.0884 \pm .0702$
Minnesota winter wheats	43	$.3238 \pm .0921$
Pure strains:		
North Dakota spring wheats	56	$-.0942 \pm .0893$
Minnesota spring wheats	48	$.1255 \pm .0958$
Wisconsin winter wheats	22	$.0601 \pm .1433$

For the nine groups of data six coefficients are positive in signs and three negative. The correlations for the Ohio winter wheats, Montana winter wheats, North Dakota spring wheats, pure strains, Minnesota spring wheats, pure strains, and Wisconsin pure strains of winter wheats are not significant, since the value of the respective coefficients is less than three times their probable error. The remaining four coefficients indicate only a very slight correlation. Of these three are positive in sign and one is negative. The highest correlation, that for the Montana

spring wheats, is based upon data from only 34 samples. From the evidence furnished by these data it would seem that so far as flour yield is concerned, and within the above range of protein content, it matters little whether the miller buys wheats of high or low protein content. This is in conformation of the results obtained by Thomas (14) who states that—

> normal, plump, dry, and sound wheat of all classes yields approximately the same percentage of flour.

PROTEIN CONTENT IN WHEAT AND LOAF VOLUME

From a commercial point of view the value of a bread wheat is determined principally by the size of loaf baked from a unit of flour of that wheat. The size of bread loaf is generally measured by the volume, expressed in cubic centimeters, of the loaf baked from a unit of flour, usually 340 gm. The volume of bread loaf is at present the only reliable index of flour strength. It is, therefore, not surprising to find that various chemical characters have been studied in an effort to establish a relationship between them and all the important factor of flour strength.

The following data are intended to express the relationship, in the light of the available material, between the volume of bread loaf and the protein in the wheat, protein in the flour, dry gluten, wet gluten gliadin, and water absorption.

TABLE IX.—*Correlation between protein in the wheat and loaf volume from Ohio winter wheats of commercial varieties*

Protein in wheat (per cent).	Loaf volume (cc.) 1,600 to 1,700	1,700	1,800	1,900	2,000	2,100	2,200	2,300 to 2,400	Total.
8.00 to 8.50	......	......	......	......	......	......	......	......	......
8.50	1	......	3	1	......	1	......	......	6
9.00	1	......	3	4	2	......	......	......	10
9.50	......	2	3	1	2	......	......	......	8
10.00	......	......	2	3	1	......	......	......	6
10.50	1	1	2	1	3	1	1	......	10
11.00	......	......	3	5	6	2	......	......	16
11.50	......	......	2	4	7	2	2	......	17
12.00	......	......	......	3	3	1	......	......	7
12.50	1	2	......	......	2	3	......	......	8
13.00	......	......	1	......	......	2	......	......	3
13.50	......	......	......	......	1	1	2	......	4
14.00	......	......	......	......	......	1	......	......	1
14.50	......	......	......	......	......	1	......	......	1
15.00	......	......	......	......	......	......	1	......	1
15.50	......	......	......	......	......	......	......	1	1
16.00	......	......	......	......	......	......	......	......	......
16.50	......	......	......	......	......	......	......	......	......
17.00	......	......	......	......	......	......	......	1	1
17.50 to 18.00	......	......	......	......	......	......	......	......	......
Total	4	5	19	22	27	15	6	2	100

$r = 0.5394 \pm 0.0479$.

TABLE X.—*Correlation coefficients for protein in wheat and loaf volume*

Kind of wheat.	Number of samples.	Coefficients of correlation.
Commercial varieties:		
North Dakota spring wheats	128	-0.1172 ± 0.0588
Minnesota spring wheats	202	$.1827 \pm .0459$
Montana spring wheats	34	$.3555 \pm .1011$
Colorado spring wheats	48	$.4908 \pm .0739$
Montana winter wheats	91	$.3638 \pm .0613$
Ohio winter wheats	100	$.5394 \pm .0479$
Minnesota winter wheats	43	$.5874 \pm .0674$
Kansas winter wheats	42	$.7547 \pm .0448$
Pure strains:		
Minnesota spring wheats	48	$.4621 \pm .0766$
Wisconsin spring wheats	22	$.5123 \pm .1061$
Maine spring wheats	31	$.5194 \pm .0885$
Ohio winter wheats	25	$.5548 \pm .0934$

The data in Tables IX and X, with the exception of those for North Dakota spring wheats and Minnesota commercial varieties of spring wheats, indicate a high, positive correlation. The coefficients of correlation range from -0.1172 ± 0.0588 for the North Dakota wheats to 0.7547 ± 0.0448 for the Kansas wheats. The negative correlation for the North Dakota wheats is not significant, since the value of the coefficient is less than three times its probable error. The coefficient for the Minnesota commercial varieties of spring wheats indicates only a slight, positive correlation. This is rather of interest since both States are centers of production of the hard red spring wheat. In this connection the observations of Thomas (*14*) may be cited which seem to throw some light on this point. Thomas found that high crude-protein content as a rule is accompanied by high strength but that the relation between these two factors varies with different classes of wheat, and extremely high crude-protein—over 15 per cent—is sometimes accompanied by a decrease in baking strength. The writer had occasion to observe several similar instances in pure lines selected from Preston wheats. Thomas further found that a wider variation in volume is noticeable with all classes of wheat for the lower percentages of protein than for the higher percentages, many samples with very high protein content being lower in strength than those having a medium content. In the light of these observations one may assume that the large number of samples with very high protein entering into the material from which the North Dakota and Minnesota data have been secured may have counteracted and neutralized the influence of the samples having a medium protein content. It should be noted, however, that the coefficients for the Minnesota pure strains of spring wheat indicate a rather high positive correlation.

PROTEIN IN FLOUR AND LOAF VOLUME

The high correlation between the protein content in the wheat and protein content in flour points to a correlation between the protein content in flour and loaf volume which will closely agree with that between the protein content in wheat and loaf volume.

TABLE XI.—*Correlation between protein in flour and loaf volume from Ohio winter wheats of commercial varieties*

VOLUME LOAF (cc.)

Protein in flour (per cent)	1,600 to 1,700	1,700	1,800	1,900	2,000	2,100	2,200	2,300 to 2,400	Total.
6.00 to 6.50	......	......	......	......	......	......	......	......	......
6.50	1	......	3	1	2	......	......	......	7
7.00	1	......	4	3	1	1	......	......	10
7.50	......	1	3	2	2	......	......	......	8
8.00	......	......	......	3	1	1	1	......	6
8.50	1	1	3	1	3	......	1	......	10
9.00	......	1	1	3	3	2	......	......	10
9.50	......	......	2	4	......	3	1	......	10
10.00	......	1	1	2	11	......	......	......	15
10.50	......	1	2	......	1	3	......	......	7
11.00	......	......	......	2	2	3	1	......	8
11.50	1	......	......	......	1	......	......	......	2
12.00	......	......	......	......	......	......	1	......	1
12.50	......	......	......	......	......	1	......	......	1
13.00	......	......	......	......	......	1	......	......	1
13.50	......	......	......	......	......	......	1	1	2
14.00	......	......	......	......	......	......	......	......	......
14.50	......	......	......	......	......	......	......	......	......
15.00	......	......	......	......	......	......	......	......	......
15.50	......	......	......	......	......	......	......	1	1
16.00 to 16.50	......	......	......	......	......	......	......	......	......
Total	4	5	19	21	27	15	6	2	99

$r=0.4709\pm0.0528$.

TABLE XII.—*Correlation coefficients for protein in flour and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation
Commercial varieties:		
North Dakota spring wheats	119	-0.0987 ± 0.0612
Minnesota spring wheats	203	$.2586\pm.0442$
Montana spring wheats	34	$.3448\pm.1019$
Colorado spring wheats	48	$.6130\pm.0608$
Montana winter wheats	91	$.3620\pm.0614$
Ohio winter wheats	99	$.4709\pm.0528$
Minnesota winter wheats	43	$.6496\pm.0594$
Kansas winter wheats	43	$.7956\pm.0377$
Pure strains:		
North Dakota spring wheats	28	$.3018\pm.1158$
Minnesota spring wheats	48	$.5469\pm.0689$
Ontario spring wheats	16	$.5752\pm.1129$
Wisconsin winter wheats	25	$.3990\pm.1134$
Ohio winter wheats	25	$.5560\pm.0932$

From these data, with one exception, the positive and generally high correlation between the loaf volume and protein in flour is evident. The correlation for the North Dakota spring wheats, commercial varieties, is again negative and without significance. An inspection of Tables X and XII further shows that the relative rank of values of the correlation coefficients is identical for both pairs of characters considered. A comparison of the absolute values of correlation coefficients for the two pairs

of variables reveals the fact, with few exceptions, the degree of correlation between loaf volume and protein in flour is higher than that between loaf volume and protein in wheat. The Ohio and Montana winter wheats constitute an exception to the general condition as indicated by the foregoing data. For the Ohio winter wheats, commercial varieties, the correlation between loaf volume and protein in flour is lower than that between loaf volume and protein in wheat (Tables IX and XI), while for the Montana winter wheats and Ohio winter wheats, pure strains, the degree of correlation between these two pairs of variables is practically identical.

DRY GLUTEN AND LOAF VOLUME

It is regretted that the data for this important pair of variables, both as to the number of wheat groups and number of samples within the groups, should be so small.

TABLE XIII.—*Correlation between dry gluten and loaf volume from Ohio winter wheats of commercial varieties*

Dry gluten (per cent).	Loaf volume (cc.).								Total.
	1,500 to 1,600	1,600	1,700	1,800	1,900	2,000	2,100	2,200 to 2,300	
6.50 to 7.00									
7.00				4	1				5
7.50	1	1		1	1	2			6
8.00		1	1	3	1	2			8
8.50		1	2	2	3	1			9
9.00				3	3	5			11
9.50				3	4	1	2		10
10.00				1	2	4			7
10.50					1	3		1	5
11.00									
11.50									
12.00						1			1
12.50						1			1
13.00 to 13.50									
Total	1	3	3	17	16	20	2	1	63

$r = 0.4770 \pm 0.0656$.

TABLE XIV.—*Correlation coefficients for dry gluten and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	37	0.0391 ± 0.1107
Colorado spring wheats	48	$.7412 \pm .0439$
Ohio winter wheats	63	$.4770 \pm .0656$
Pure strains:		
Maine spring wheats	31	$.5797 \pm .0806$
Ontario spring wheats	16	$.6078 \pm .1063$

The data in Tables XIII and XIV indicate, again with one exception, positive high correlations. The coefficients of correlation range from 0.0391 ± 0.1107 for the North Dakota spring wheats to 0.7412 ± 0.0439

for the Colorado spring wheats. The degree of correlation between dry gluten content and loaf volume for the Ohio winter wheats is practically identical with that between protein in flour and loaf volume for the same data, while with the other wheats the data indicate a higher correlation between gluten and loaf volume than that between protein in flour and loaf volume. The correlation for the North Dakota spring wheats is not significant.

WET GLUTEN AND LOAF VOLUME

What has been said about the paucity of data for the characters dry gluten and loaf volume holds also for the data on wet gluten and loaf volume.

TABLE XV.—*Correlation between wet gluten and loaf volume from Colorado spring wheats of commercial varieties*

Wet gluten (per cent).	Loaf volume (cc.)								Total.
	1,000 to 1,100	1,100	1,200	1,300	1,400	1,500	1,600	1,700 to 1,800	
21.00 to 23.00									
23.00				1	1				2
25.00				4	2	1			7
27.00			1			1			2
29.00					3	1			4
31.00		2		1	5	4			12
33.00					3	3	1		7
35.00	1					3	1		5
37.00					1	1			2
39.00						1	1		2
41.00						1			1
43.00							1		1
45.00									
47.00							1	1	2
49.00								1	1
51.00 to 53.00									
Total	1	2	1	6	15	16	5	2	48

$r = 0.5025 \pm 0.0728$.

TABLE XVI.—*Correlation coefficients for wet gluten and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	37	0.2600 ± 0.1034
Colorado spring wheats	48	.5025 ± .0728
Ohio winter wheats	29	.5425 ± .0883
Pure strains:		
Maine spring wheats	31	.4573 ± .0958
Ontario spring wheats	16	.6062 ± .1067

The coefficients for the North Dakota spring wheats and Ohio winter wheats show a higher correlation between the characters here considered than that between dry gluten and loaf volume. The correlation for the North Dakota wheats, however, is not significant. The correlation

between wet gluten and loaf volume for the Colorado and Maine wheats is lower than that between dry gluten and loaf volume, while the degree of relationship of the two pairs of variables is almost identical for the Ontario wheats.

WATER ABSORPTION AND LOAF VOLUME

Strong flours are regarded as being possessed of a higher capacity for water absorption than weak flours. From this a positive correlation between water absorption and loaf volume is to be expected.

TABLE XVII.—*Correlation between water absorption and loaf volume from Minnesota spring wheats of commercial varieties*

Water absorption (per cent).	Loaf volume (cc.).								Total.
	2,000 to 2,100	2,100	2,200	2,300	2,400	2,500	2,600	2,700 to 2,800	
47 to 48	.....	.....	.....	.....	.....	.....	.....	.....	.....
48	2	.....	.....	.....	.....	.....	.....	.....	2
49	.....	.....	.....	.....	1	.....	.....	.....	1
50	1	4	1	.....	2	.....	.....	.....	8
51	.....	.....	.....	.....	2	.....	.....	.....	2
52	.....	1	2	.....	1	2	.....	.....	6
53	.....	.....	.....	.....	2	2	2	.....	6
54	.....	.....	2	.....	1	4	2	1	10
55	.....	1	2	1	.....	.....	2	.....	6
56	.....	1	1	2	1	6	3	.....	14
57	.....	2	.....	4	7	5	5	.....	23
58	.....	.....	2	1	3	7	4	.....	17
59	.....	.....	1	1	8	20	3	.....	33
60	.....	.....	1	1	9	12	.....	2	25
61	1	.....	.....	1	3	12	2	.....	19
62	.....	.....	.....	.....	3	8	1	.....	12
63	.....	.....	.....	1	5	4	2	.....	12
64	.....	.....	.....	.....	1	1	.....	.....	2
65	.....	.....	.....	.....	1	1	1	.....	3
66 to 67	.....	.....	.....	.....	.....	.....	.....	.....	.....
Total	4	9	12	12	50	84	27	3	201

$r = 0.3760 \pm 0.0408$.

TABLE XVIII.—*Correlation coefficients for water absorption and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation
Commercial varieties:		
North Dakota spring wheats	119	0.2099±0.0591
Montana spring wheats	34	.2546± .1082
Minnesota spring wheats	201	.3760± .0408
Montana winter wheats	91	.0348± .0706
Minnesota winter wheats	43	.4765± .0795
Ohio winter wheats	86	.5291± .0524
Kansas winter wheats	42	.6601± .0587
Pure strains:		
Minnesota spring wheats	48	.0100± .0973
North Dakota spring wheats	27	.4116± .1078
Ohio winter wheats	31	.1498± .1184

Of the 10 coefficients of correlation given in Tables XVII and XVIII, 3—those for Montana winter wheats, Minnesota spring wheats, pure strains, and Ohio winter wheats, pure strains—indicate no significant correlation, since their value is considerably less than three times their probable error. The correlation for the winter wheats are, with two exceptions, higher than for the spring wheats.

FLOUR YIELD AND LOAF VOLUME

The coefficients of correlation for this pair of variables do not indicate a consistent trend. Of the eight coefficients, four are positive in sign and four negative. Five coefficients indicate virtually no correlation, as their value is less than three times their probable error. Of the remaining three coefficients, two indicate only a slight positive correlation for the Minnesota, winter, and spring wheats, and one the highest but negative correlation, -0.3920 ± 0.0763 for the North Dakota spring wheats, pure lines. In considering these data the influence of the condition of wheat upon the flour yield should be borne in mind, the shrunken wheat often yielding less flour and a larger volume of bread loaf than very plump, sound wheat.

TABLE XIX.—*Correlation between yield of flour and loaf volume from Minnesota spring wheats of commercial varieties*

Yield of flour (per cent).	Loaf volume (cc.).								Total.
	2,000 to 2,100	2,100	2,200	2,300	2,400	2,500	2,600	2,700 to 2,800	
64-65	.....	.....	.....	.....	.....	.....	.....	.....	.....
65	.....	.....	1	.....	2	.....	.....	.....	3
66	.....	.....	1	2	.....	.....	.....	.....	3
67	.....	.....	.....	1	2	5	.....	.....	8
68	.....	2	.....	.....	1	2	.....	.....	5
69	1	.....	2	3	2	11	5	.....	24
70	.....	2	5	1	14	12	4	.....	38
71	.....	2	.....	2	7	16	5	.....	32
72	.....	1	1	2	4	18	1	.....	27
73	3	2	1	1	13	15	10	2	47
74	.....	.....	.....	.....	2	4	1	1	8
75	.....	.....	1	.....	3	.....	1	.....	5
76	.....	.....	.....	.....	.....	2	.....	.....	2
77-78	.....	.....	.....	.....	.....	.....	.....	.....	.....
Total	4	9	12	12	50	85	27	3	202

$r = 0.1524 \pm 0.0464$.

TABLE XX.—*Correlation coefficients for yield of flour and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	99	−0.0900±0.0672
Montana spring wheats	34	.1425± .1133
Minnesota spring wheats	202	.1524± .0464
Montana winter wheats	91	−.0530± .0705
Ohio winter wheats	29	−.1440± .1227
Minnesota winter wheats	43	.3392± .0910
Pure strains:		
North Dakota spring wheats	56	−.3920± .0763
Minnesota spring wheats	48	.1283± .0958

GLIADIN AND LOAF VOLUME

As already stated, the gliadin content has been suggested as a factor determining strength in wheat flour. In Table XXI are given data indicating the relationship between gliadin and strength as measured by loaf volume. The correlation for the North Dakota wheats, commercial varieties, is again negative, that for the Colorado is positive, but both are hardly significant as their value is less than three times their probable error. The remaining correlations are positive and some of them very high, ranging from 0.3815±0.0960 for the Kansas wheats to 0.7455±0.0598 for the North Dakota spring wheats, pure strains.

TABLE XXI.—*Correlation coefficients for gliadin and loaf volume*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	113	−0.1718±0.0616
Colorado spring wheats	48	.0308± .0973
Kansas winter wheats	36	.3815± .0960
Pure strains:		
Ontario spring wheats	16	.6304± .1017
North Dakota spring wheats	25	.7455± .0598

DRY GLUTEN AND GLIADIN

Gliadin is one of the constituents of gluten, and a close association between these two values is to be expected. The data in Tables XXII and XXIII are rather meager and indicate a positive correlation, the coefficients ranging from 0.3941±0.1058 for the Ohio wheats, which is rather low, to 0.9352±0.0212 for the Ontario wheats.

TABLE XXII.—*Correlation between dry gluten and gliadin content of North Dakota spring wheats of commercial varieties*

Dry gluten (per cent).	Gliadin (per cent).											Total.
	4.00 to 4.50	4.50	5.00	5.50	6.00	6.50	7.00	7.50	8.00	8.50	9.00 to 9.50	
9.00 to 9.50	....	....	....	....	....	....	....	....	....	....	....	...
9.50	....	....	....	....	....	....	1	....	....	....	....	1
10.00	....	....	....	....	....	....	....	....	....	....	....	...
10.50	....	....	....	....	....	....	....	....	....	....	....	...
11.00	....	....	....	1	....	....	....	....	....	....	....	1
11.50	....	....	....	....	....	....	....	....	....	....	....	...
12.00	....	....	....	....	1	1	....	....	....	....	....	2
12.50	....	....	....	....	....	....	2	....	....	....	....	2
13.00	....	....	....	1	1	2	....	....	....	....	....	4
13.50	....	....	....	....	....	1	1	2	....	....	....	4
14.00	....	....	....	....	....	....	....	....	....	....	....	...
14.50	1	....	....	....	....	1	....	2	....	....	....	4
15.00	....	....	....	....	....	....	....	3	....	....	....	3
15.50	....	....	....	....	....	....	1	....	3	1	....	5
16.00	....	....	....	....	....	....	1	....	....	2	....	3
16.50	....	....	....	....	....	....	....	....	....	....	1	1
17.00	....	....	....	....	....	....	....	....	....	1	1	2
17.50	....	....	....	....	....	....	1	....	....	....	....	1
18.00	....	....	....	....	....	....	....	1	....	....	....	1
18.50	....	....	....	....	....	....	....	....	....	....	1	1
19.00	....	....	....	....	....	....	....	....	1	....	....	1
19.50 to 20.00	....	....	....	....	....	....	....	....	....	....	....	...
Total	1	....	....	2	2	5	7	8	4	4	3	36

$r = 0.6107 \pm .0704$.

TABLE XXIII.—*Correlation coefficients for dry gluten and gliadin content*

Kind of wheat	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	36	0.6107 ± 0.0704
Ohio winter wheats	29	$.3941 \pm .1058$
Pure strains:		
Ontario spring wheats	16	$.9352 \pm .0212$

DRY GLUTEN AND WATER ABSORPTION

Since high gluten content and water absorption are generally associated with high baking strength as measured by loaf volume (Tables XIII, XIV, XVII, and XVIII) one might expect some relationship between dry gluten content and water absorption.

TABLE XXIV.—*Correlation between dry gluten content and water absorption in Ohio winter wheats of commercial varieties*

Dry gluten (per cent).	Water absorption (per cent).													Total.
	56 to 57	57	58	59	60	61	62	63	64	65	66	67	68 to 69	
6.50 to 7.00	....	....	....	....	....	....	....	....	....	....	....	....	....	....
7.00	....	1	....	....	1	2	1	....	....	....	....	....	....	5
7.50	....	1	1	1	1	....	1	1	....	....	....	....	....	6
8.00	....	1	3	....	1	2	1	....	....	....	....	....	....	8
8.50	2	2	1	....	2	....	....	....	1	....	....	....	1	9
9.00	....	1	3	1	3	2	1	....	....	....	....	....	....	11
9.50	....	1	....	1	4	2	....	....	1	1	....	....	....	10
10.00	....	....	....	....	2	5	....	....	....	....	....	....	....	7
10.50	....	1	....	....	....	1	....	2	....	....	....	....	1	5
11.00	....	....	....	....	....	....	....	....	....	....	....	....	....	....
11.50	....	....	....	....	....	....	....	....	....	....	....	....	....	....
12.00	....	....	....	....	....	....	1	....	....	....	....	....	....	1
12.50	....	....	....	....	1	....	....	....	....	....	....	....	....	1
13.00 to 13.50	....	....	....	....	....	....	....	....	....	....	....	....	....	....
Total	2	8	8	3	15	14	5	3	2	1	....	....	2	63

$r = 0.2211 \pm 0.0808$.

TABLE XXV.—*Correlation coefficients for dry gluten content and water absorption*

Kind of wheat.	Number of samples.	Coefficient of correlation.
Commercial varieties:		
North Dakota spring wheats	37	0.1406±0.1087
Ohio winter wheats	63	.2211± .0808
Pure strains:		
Ohio winter wheats	19	−.1319± .1520

The data in Tables XXIV and XXV do not bear out this expectation, since they indicate hardly any significant correlation. An interpretation of these data would lead to the conclusion that flours with high gluten content behave indifferently in regard to water absorptive capacity. It is only fair, however, to note the small number of samples from which these correlation coefficients have been computed. Caution should be exercised in interpreting the value of the correlation coefficients as indicated by the probable errors in cases where the number of samples is small.

QUALITY OF GLUTEN AND LOAF VOLUME

Practical experience in the bakehouse shows that strength in wheat flour is best reflected by the quality of its gluten. The measuring of the intensity of the association between these two characters can not be effected by computing the coefficient of correlation, since one of these variables, quality, can not be numerically defined but is only estimated in descriptive terms. Accordingly, as a measure of the degree of association between gluten quality and loaf volume the correlation ratio,

η, has been computed. Eight degrees of gluten quality have here been considered ranging from very soft and sticky, dead, nonelastic gluten to very good, strong, very elastic. In Table XXVI are given the correlation ratios for three groups of wheat.

TABLE XXVI.—*Correlation ratio, η, for quality of gluten and loaf volume*

Kind of wheat.	Number of samples.	Correlation ratio.
Pure strains:		
Maine spring wheats	31	0.7418±0.0544
Ontario spring wheats	16	[a] .9478± .0171
Wisconsin winter wheats	16	.7456± .0748

[a] Because of its small number of observations the regression for this table is not linear. The value of the coefficient is too high. The regression lines for the other two tables appear to be linear.

From these data the very high and consistent relationship between gluten quality and loaf volume will be noted. Notwithstanding the paucity of data and the fact that the constant η generally has a somewhat greater value for the same data than the coefficient of correlation, it is safe to conclude that loaf volume is more closely associated with gluten quality than with any other character considered. While it is no doubt true that the character quality of gluten is determined by estimation and hence may be subject to variation due to personal equation, yet the determinations of gluten quality made according to a definite standard in a given laboratory may be regarded as fairly constant.

DRY GLUTEN CONTENT AND QUALITY OF GLUTEN

In considering the results of a chemical analysis of flours from pure lines of wheat grown in Aroostook, the writer (18, p. 37) noticed that while high gluten content in flours from pure wheat strains selected from a given commercial variety was not generally associated with good quality of gluten, yet the flours from pure strains of the same variety showing a low gluten content were generally also low in gluten quality. The degree of relationship between gluten content and gluten quality as measured by the constant η is given in the data in Table XXVII.

TABLE XXVII.—*Correlation ratio, η, for dry gluten content and quality of gluten*

Kind of wheat.	Number of samples.	Correlation ratio.
Pure strains:		
Maine spring wheats	31	0.5994±0.0777
Ontario spring wheats	16	[a] .6565± .0958
Wisconsin winter wheats	16	.5155± .1237

[a] As in Table XXIV, the regression lines for the Ontario spring wheats are not linear, the value of η comes chiefly from the zigzag nature of the regression lines and not from any true relationship between the variables. The lines for the Maine and Wisconsin wheats appear to be linear.

The number of flour samples for which these two variables have been determined is small, but the data generally indicate a fairly high correlation between quantity and quality of gluten. This relationship accounts for the frequently recorded observation that glutenous wheats are strong

and furnish a larger loaf volume from a unit of flour than wheats with a low gluten content. The relationship is by no means perfect, probably because of the fact that a number of wheats with high gluten content fail to show good quality of gluten. In this connection it should be noted that the relationship between these two variables is to be studied for pure strains belonging to a given variety or group of wheat and considered within that group since there is some evidence indicating that certain wheat groups carry a larger number of varieties or strains of very high gluten content of poor quality than others. The durum and Preston wheats may be cited as illustrating this point.

DISCUSSION OF RESULTS

The relations considered in the preceding section will now briefly be considered from the standpoint of the plant breeder who desires to make use of any correlation between a wheat character determinable at an early stage of the pure line selection work and the baking strength of flour.

Tables I to VI show data expressing the relationship between the crude protein in the wheat and its constituents, protein in flour, gluten, and gliadin. The correlations are very high and consistent, as would be expected. The correlation between crude protein content in the wheat and that in flour is so high that, for practical purposes, the value for the protein content in the flour may be substituted for that in the wheat. This would eliminate the necessity of securing a sample of flour for analysis, which requires a larger amount of grain and an experimental mill to grind it, whereas for the determination of crude protein in the grain the yield of a single plant is sufficient.

Of great interest are the correlations between loaf volume and the other chemical characters. The data given in Tables IX to XII indicate that the relation between loaf volume and crude protein in the wheat is paralleled by that between loaf volume and protein in the flour, the difference being one of degree as the relationship for the latter pair of characters is of greater intensity than that for the former. Since the correlation between loaf volume and protein in the flour parallels that between loaf volume and crude protein in the wheat, it would follow that, again for practical purposes, it may be sufficient to consider the crude protein in the wheat alone in relationship to strength of flour.

Considering the gluten content and loaf volume, the data in Tables XIII and XIV, which are rather scant, indicate a very high, positive correlation between these two variables. Gliadin, which is one of the constituents of gluten and closely correlated with it (Tables XII and XXIII), generally bears a close relation to loaf volume (Table XXI).

Finally, considering the quality of gluten and loaf volume, it will be seen from Tables XXVI and XXVII that loaf volume is more closely correlated with gluten quality than with any other character under consideration.

From this brief consideration of the more important correlations it should be clear that as far as strength of flour is concerned gluten content and gluten quality show the highest degree of association with loaf volume. If these correlations are accepted as a working basis, the problem of finding a practical index of strength of flour is reduced to the task of determining the gluten content and gluten quality. In order to determine these two values a certain amount of flour is required. This is not avail-

ible until after the propagation of the pure strains of wheat has reached a certain stage. In order to judge the gluten quality, when only a very small sample of grain is available, say the yield of a single plant, or even of a spike, use may be made of a rather crude but fairly accurate method, the chewing test. The chewing test gives also some approximate idea as to gluten content. The gluten content well reflects, in fact, parallels the crude protein content in the wheat. Furthermore, both the gluten content and crude protein content are highly correlated with loaf volume. From this it would follow that the crude protein content in the wheat may, for practical purposes, be substituted for gluten content. Thus the crude protein content in the wheat and the chewing test may be accepted as a practical working index of strength in wheat in the early stages of the selection work when the plant breeder is confronted with the task of deciding which of the numerous strains to retain and which to discard. The information afforded by this index may further be supplemented by examining the grain for color and hardness.

The determination of the crude protein content in the grain of a given strain gains in importance and value in view of the accumulating evidence which indicates that while the absolute amount of protein in wheat may be influenced by environment, yet the protein content of wheat is a varietal characteristic, and moreover, that there is a tendency for the different varieties and strains to retain their relative rank with respect to protein content from one year to the next (*18*).

SUMMARY

The present paper is the result of a study of the relationship of the different characters of wheat based upon published data obtained from analysis of a number of American wheats.

Subject to the limitations of the material which have been pointed out in this paper, the following conclusions may be drawn from the data herein presented:

Crude protein content in the wheat is very closely and consistently correlated with protein in flour, dry gluten, and gliadin.

There appears to be practically no relation between crude protein content in the wheat and flour yield.

There is, with some notable exceptions, a high positive correlation between the crude protein in the wheat and strength of flour as determined by the loaf volume.

There is generally even a higher, positive correlation between protein in the flour and loaf volume.

The gluten content of the flour is very closely correlated with loaf volume. The intensity of association between these two variables appears to be greater than that for protein in flour and loaf volume.

There is generally a high, positive correlation between wet gluten content and loaf volume.

There is, with some exceptions, a positive, fairly high correlation between water absorption of the dough and loaf volume.

In normal, sound wheat there is apparently no significant correlation between flour yield and loaf volume.

Excepting the data for a few wheat groups, there is a positive and, for some wheat groups, a very high correlation between gliadin content and loaf volume.

Dry gluten content is generally highly correlated with gliadin content.

From the scant data for dry gluten content and water absorption it would seem that there is no significant correlation between these two variables, which is contrary to expectation.

Loaf volume is more closely associated with gluten quality as indicated by the correlation ratio than with any other character considered.

There appears to be a fairly high correlation between the content and quality of gluten.

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APLASTOMORPHA VANDINEI TUCKER, AN IMPORTANT PARASITE OF SITOPHILUS ORYZA L.[1]

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INTRODUCTION

The most effective insect enemy of the rice weevil, *Sitophilus oryza* L., in the United States is the chalcid *Aplastomorpha vandinei* Tucker.

While engaged in studying the biology of the rice weevil at Orlando, Fla., during the years 1920 and 1921, the writer made the following observations on the life history and habits of this parasite.

ORIGIN AND DISTRIBUTION

Although originally described as from Texas, the native home of this chalcid is unknown. It is now cosmopolitan in its distribution, probably occurring wherever corn is used.

HISTORY AND SYNONYMY

The earliest definite reference to this species in literature appeared in 1899, when Chittenden (*2*)[2] recorded it as parasitic on *Bruchus quadrimaculatus* Fab. under the name of *Apalastomorpha prattii* Ashm. MSS.

No description of this species was published, however, until 1910, when Tucker (*10*) described it under the name of *Meraporus vandinei* from Llano, Tex.

In 1913 the same species was described by Crawford (*3*) as *Aplastomorpha pratti*, the manuscript name previously given by Ashmead to individuals of this species bred in Washington, D. C., from *Bruchus quadrimaculatus* Fab. Crawford's description was based on specimens collected in Dallas, Tex., by W. D. Hunter.

Later in the same year the species was again described, this time by Girault (*5*), under the name *Neocatolaccus australiensis*, from material reared in Australia.

In 1915 Girault (*6*) placed his species *australiensis* in the genus Aplastomorpha Crawford and in 1917 (*7*) reduced *Aplastomorpha pratti* Craw., *A. australiensis* Gir., and *Meraporus vandinei* Tucker to synonymy with *Neocatolaccus vandinei* Tucker.

Finally Gahan (*4*) in 1920 recognized *Aplastomorpha vandinei* Tucker as the correct name of the species.

ECONOMIC IMPORTANCE

Aplastomorpha vandinei is of chief economic importance as a parasite of the grain weevils *Sitophilus oryza* and *S. granarius* L. It has also been reported as parasitic on several other insects of economic importance—that is, on the 4-spotted bean weevil, *Bruchus quadrimaculatus*, by Chittenden (*2*), on the cigarette beetle, *Lasioderma serricorne* Fab., by Runner (*9*) and Bodkin (*1*), on *Pachymerus* sp. by Girault (*8*), and on the "grain

[1] Accepted for publication May 13, 1922.
[2] Reference is made by number (italic) to "Literature cited," p. 556.

moth" and miscellaneous forest galls by Girault (5). The writer has bred it from *Sitophilus oryza*, *S. granarius*, and also from the broad-nosed grain weevil, *Caulophilus latinasus* Say.

DESCRIPTION OF THE SPECIES

The female (Pl. 1, A) is normally about 2 mm. in length, of characteristic chalcid form, dark metallic green in color, with yellow legs. The male (Pl. 1, G) is somewhat smaller and differs from the female in having the abdomen brown, with a large yellowish white spot. Both sexes vary considerably in size.

TECHNICAL DESCRIPTION (*10*)

Female: Head and thorax dark greenish, clothed with thin and fine silvery pubescence, a thick linear patch on each side of the metathorax; abdomen smooth, shining greenish, thinly pubescent on apical segments, venter deeply keeled; head transverse, somewhat wider than thorax, finely reticulated rather than punctured on occiput, front and cheeks with convergent striæ at oral margin of middle of face; front above insertion of antennæ hollowed for reception of scapes; mouth-parts ferruginous, each mandible with four denticles; antennæ about as long as thorax, scape dull reddish; flagellum dull reddish beneath, darker above, with fine silvery pubescence; pedicel about twice the length of the three ring joints together, but scarcely longer than the first funicle joint, which appears distinctly longer than wide, second and third funicle joints slightly longer than wide, fourth and fifth quadrate; club expanded at junction of first and second joints, the third forming a small conical tip. Anterior ocellus situated but little in advance of a median point between the posterior ones.

Thorax with fine shallow thimble-pitted punctures, contiguous and distinctly larger than on head; length of thorax scarcely exceeds the width, parapsidal furrows very faint only on anterior half of mesonotum. Metathorax very finely punctured, with a median longitudinal carina; metathoracic neck very short, smooth; lateral folds indicated by basal foveolæ only, spiracles very small, broadly oval; spiracular sulci very deep and distinct.

Tegulæ fulvous; wing-veins yellow, ciliate, stigmal vein shorter than marginal or postmarginal by about one-fourth the length, legs yellow, excepting the coxæ, femora in greater part between the base and apex, and last tarsal joint outwardly, which are dark brown or fuliginous. Tibiæ of middle and posterior legs little longer than femora or tarsi, which are about equal in length, but no noticeable difference in these respects with fore legs. Comparatively the fore legs are shorter than the others. [Length ranges from 1.25 mm. to 2 mm.]

Male: Antennæ with larger microscopical pits than female; the first and second ring joints very small and compressed, the third appears as the first joint of the funicle, but is smaller and shorter than the true funicular joints. Abdomen hardly as long, or at most not longer than thorax, almost flattened above, not deeply keeled ventrally, widest near apex, and with a dorsal fulvous area near base expanding to the lateral edges; otherwise agreeing with female. [Length ranges from 1 mm. to 1.5 mm.]

METHODS USED IN STUDY OF PARASITES

The immature stages of the rice weevil, *Sitophilus oryza*, which served as a host for the parasite in this study, are passed entirely concealed within the seeds of some of our common grains; hence of necessity the immature stages of the attacking parasite are also concealed from sight within the recesses of the grain that harbors the weevil. In order to observe the method of attack and the subsequent development of the parasite the following method was devised: Larvæ of the rice weevil were placed singly in small gelatin capsules, a small piece of absorbent cotton being packed in behind them to hold them in place. The capsules thus loaded were then placed in glass vials containing parasites.

The parasites in every instance accepted the loaded capsules in lieu of infested grain, located the weevil larvæ, pierced the shell of the capsules with their slender ovipositors, stung the larvæ into quiescence, and deposited their eggs.

Since the gelatin capsules were transparent, every phase of the attack and oviposition was clearly visible. After oviposition had taken place the capsules were removed and kept for observation. The eggs hatched and the young parasitic grubs developed to maturity in a perfectly normal manner, all changes being readily observed.

In addition to being able to observe the development of the parasites it was a simple matter to count the number of eggs deposited daily by individual parasites.

It was found that by pricking small holes through the shell of the capsules in the vicinity of the enclosed larvæ, the parasites would avail themselves of the holes to attack the larvæ and deposit eggs instead of going to the trouble of boring through the gelatin.

The parasites were fed on a sirup made from sugar and water.

LIFE HISTORY OF PARASITE

COPULATION

Copulation normally takes place shortly after emergence. Sex attraction is weak, the male showing no evidence of interest except when in close proximity to the female. In captivity the female more often seeks the male at first, moving around in the proximity of the male and stopping to preen her wings and body. After becoming conscious of her presence the male evinces signs of excitement, follows her about, and springs to her back. He then caresses antennæ with her for a brief period before copulating. In the act of copulation the female tilts her abdomen, and the abdomen of the male is curved down at one side of or directly over the end of the abdomen of the female while he clings to her wings and body. Copulation lasts for from 5 to 35 seconds.

OVIPOSITION

Oviposition usually begins on the first or second day after the emergence of the female, although it may begin the day of emergence. The stage of the host attacked is apparently of no great moment as long as it is large enough to permit the parasitic grub to develop to maturity. The fourth larval, prepupal, and pupal stages are all attacked, though the preference seems to lie with the larval stage.

The female parasite crawls around over the infested grain, exploring carefully the surface of the grain with the tips of her antennæ. Possibly the movements of the feeding weevil grub are detected by the sensitive tips of the antennæ; at any rate, the position of the grub is soon located. The ovipositor is brought into position with the tip of the abdomen, thrust through the shell of the grain, and plunged into the weevil grub. The ovipositor is apparently whipped around just under the skin until the grub is reduced to a paralyzed or quiescent state. The ovipositor is then withdrawn. It is again thrust through the shell of the grain and an egg is deposited on the exterior of the grub or in close proximity to it.

Only one egg is deposited at a time and usually but one egg is deposited with each larva or pupa attacked. With the parasites under observation it was not uncommon for two, three, four, or even more eggs to be deposited on one larva, but this was probably due in part to the effects of confinement and the limited supply of host material.

When several eggs are deposited on a single host grub it is rarely that more than one parasite reaches maturity; the writer has, however, observed a few instances where two parasites were reared on the same host.

OVIPOSITION PERIOD

The oviposition period extends over the greater part of the life of the female parasite. Commencing a day or two after emergence it lasts to within a week or two before death. The longest oviposition period recorded was 73 days, from December 4, 1920, to February 14, 1921, the shortest 39 days, from August 2, 1920, to September 9, 1920.

Eggs are laid at the rate of from 1 to 12 a day, depending upon the temperature. During the summer months the oviposition rate is high, but in winter the rate decreases to 1 or 2 a day. On the colder days oviposition ceases entirely.

NUMBER OF EGGS LAID

The greatest number of eggs laid by a single female was 283. These were laid in the months of August and September, 1920, over a period of 43 days. The lowest number recorded was 51. These were laid during the months of December, 1920, and January and February, 1921, over a period of 73 days. In general more eggs are laid by the summer generations of parasites than by those of winter.

Table I contains data concerning the oviposition period, the number of eggs laid, longevity, etc.

TABLE I.—*Data concerning oviposition and longevity of Aplastomorpha vandinei at Orlando, Fla., 1920–21*

No.	Date parasite emerged.	Date first egg was laid.	Length of preoviposition period.	Date last egg was laid.	Length of oviposition period.	Number of eggs laid.	Date of death.	Length of life.
			Days.		*Days.*			*Days.*
1	July 28	July 29	1	Sept. 9	43	283	Sept. 10	45
2	July 29	July 30	1	Sept. 21	54	258	Oct. 11	75
3[1]	Aug. 11	Aug. 11	½	Sept. 29	50	157	Nov. 8	90
4	Aug. 17	Aug. 18	1	Oct. 24	68	236	Nov. 4	80
5	...do.....	...do.....	1	Oct. 27	71	239	Nov. 1	77
6	Aug. 20	Aug. 21	1	Oct. 30	71	279	Nov. 12	85
7	Oct. 15	Oct. 17	2	Dec. 22	67	93	Jan. 10	88
8	Nov. 1	Nov. 3	2	Jan. 12	71	52	Jan. 21	82
9	Nov. 21	Nov. 26	5	Feb. 5	72	79	Feb. 15	87
10	Dec. 2	Dec. 4	2	Feb. 14	73	51	...do.....	76

[1] Unfertilized female.

THE EGG

The egg (Pl. 1, F) is opaque shining white, the surface somewhat roughened with minute raised spots; it varies considerably in shape but is usually somewhat spindle-shaped, with one end thicker and more rounded than the other. Length 0.46 to 0.5 mm., width 0.14 to 0.16 mm.

INCUBATION PERIOD

The egg hatches in from 1½ to 2 days during the warm months of the year, but in winter the incubation period is lengthened to as many as 5 days.

The developing embryo may be seen near the center of the egg as a cloudy white oval body. As the embryo develops the contents of the egg draw away from the two ends of the tough chorion, which shrivels and wrinkles. In the process of hatching, the chorion is ruptured at the broad end of the egg and the young larva emerges.

LARVA STAGE

The newly emerged larva is about 0.5 mm. long and 0.20 mm. wide. It is a footless, fleshy grub, widest at the head and thorax and tapering caudad. It is transparent, smooth, and shining; body composed of 13 segments besides the head; head provided with a pair of slender chitinized mandibles (Pl. 1, B) 0.019 to 0.02 mm. in length.

If the egg is not attached to the host, the larva on hatching begins at once to seek for food, moving toward the host grub with an undulating movement of the body segments. Upon reaching its host the larva wanders about for a while over the grub but soon settles down to feed. It will feed on almost any part of the host.

The parasitic grub feeds rapidly and in summer becomes full grown in from 3 to 5 days. During cold weather the larval period is somewhat prolonged. Table II will give an idea of the variation in the length of this period at various times of the year.

TABLE II.—*Life-history data on Aplastomorpha vandinei*

No.	Date egg laid.	Date egg hatched.	Length of egg stage.	Date pupated.	Length of larval stage.	Date adult emerged.	Length of pupal stage.	Period from egg to adult.
			Days.		*Days.*		*Days.*	*Days.*
1	July 27	July 29	2	Aug. 3	5	Aug. 11	8	15
2	Aug. 3	Aug. 5	2	Aug. 10	5	Aug. 17	7	14
3	Aug. 5	Aug. 7	2	Aug. 12	5	Aug. 19	7	14
4	Aug. 5	Aug. 7	2	Aug. 11	4	Aug. 18	7	13
5	Aug. 6	Aug. 8	2	Aug. 12	4	Aug. 18	6	12
6	Aug. 13	Aug. 15	2	Aug. 18	3	Aug. 24	6	11
7	Aug. 13	Aug. 15	2	Aug. 18	3	Aug. 25	7	12
8	Aug. 14	Aug. 16	2	Aug. 20	4	Aug. 27	7	13
9	Aug. 15	Aug. 17	2	Aug. 21	4	Aug. 28	7	13
10	Aug. 22	Aug. 24	2	Aug. 27	3	Sept. 3	7	12
			[1] 2		[1] 4		[1] 7	[1] 13
11	Oct. 19	Oct. 21	2	Oct. 26	5	Nov. 9	14	21
12	Oct. 20	Oct. 23	3	Oct. 28	5	Nov. 10	13	21
13	Oct. 21	Oct. 23	2	Oct. 28	5	Nov. 11	14	21
14	Oct. 26	Oct. 28	2	Nov. 8	11	Nov. 23	15	28
15	Oct. 28	Oct. 30	2	Nov. 8	9	Nov. 27	19	30
16	Nov. 2	Nov. 4	2	Nov. 12	8	Dec. 7	25	35
17	Nov. 6	Nov. 8	2	Nov. 18	10	Dec. 12	24	36
18	Nov. 9	Nov. 11	2	Nov. 23	12	Dec. 28	35	49
19	Nov. 23	Nov. 25	2	Dec. 13	18	Jan. 8	26	46
20	Nov. 30	Dec. 5	5	Dec. 22	17	Jan. 19	28	50
			[2] 2.4		[2] 10		[2] 21.3	[2] 33.7
21	Dec. 2	Dec. 6	4	Dec. 23	17	Jan. 23	31	52
22	Dec. 4	Dec. 8	4	Dec. 23	15	Jan. 13	21	40
23	Dec. 5	Dec. 9	4	Dec. 27	18	Jan. 19	23	45
24	Dec. 7	Dec. 11	4	Dec. 27	16	Jan. 20	24	44
25	Dec. 8	Dec. 12	4	Dec. 28	16	Jan. 22	25	45
26	Jan. 3	Jan. 8	5	Jan. 21	13	Feb. 17	27	45
27	Jan. 5	Jan. 9	4	Jan. 27	18	Feb. 20	24	46
28	Jan. 13	Jan. 17	4	Feb. 3	17	Feb. 20	17	38
29	Jan. 14	Jan. 18	4	Feb. 5	18	Feb. 20	15	37
30	Jan. 19	Jan. 23	4	Feb. 10	18	Feb. 27	17	39
			[3] 4		[3] 16.6		[3] 22.4	[3] 43.1

[1] Summer average. [2] Fall average. [3] Winter average.

DESCRIPTION OF LARVA

The full-grown larva (Pl. 1, D) is of typical chalcid form; widest at the middle, it tapers both cephalad and caudad; smooth and shining in appearance, white in color but muddy-looking from the contents of the alimentary canal, which is greatly distended and appears nearly to fill the body cavity. The head is fairly prominent, bearing two tubercules and several small setæ, mouth-parts consisting mainly of two chitinized mandibles (Pl. 2, C) 0.053 to 0.057 mm. in length; mesothoracic, metathoracic, and first 7 abdominal segments bearing a pair of spiracles. The thoracic segments are provided with 4 setæ on each side. Abdominal segments 1 to 9, inclusive, have a subdorsal and a sublateral row of setæ on each side, 1 seta per segment in each row. The anal segment has a dorsal and ventral lobe, dorsal lobe has 4 setæ, and ventral lobe has 2 setæ. Length 2.7 to 3.0 mm., width 1.0 to 1.2 mm.

PREPUPAL STAGE

When full-grown the larva voids the entire contents of the alimentary tract. It becomes strikingly white in color and assumes a quiescent or prepupal stage. This stage normally lasts but a few hours, when the true pupal form is assumed.

PUPA

The pupa (Pl. 1, E), is at first perfectly white in color but soon turns to a pale yellowish brown, the eyes and ocelli turning reddish, and the mandibles brown. As the pupa gets older the eyes turn a darker red, and the thorax becomes dark in color followed by the head. Finally the abdomen of the male becomes banded with black and that of the female entirely black. The sexes may be readily distinguished in this stage by the difference in size, the females being considerably larger than the males. Male: Length 1.45 to 2.0 mm., width 0.61 to 0.79 mm. Female: Length 2.2 to 2.4 mm., width 0.90 to 1.0 mm.

The pupal stage lasts for a period of from 6 to 7 days. The males usually emerge in 6 days, while the females take 7. In winter the pupal stage is considerably prolonged, sometimes as long as 30 days.

HABITS OF ADULTS

In order to test the effect of light and darkness on the activity of the parasites, experiments were conducted in which corn infested with *Sitophilus oryza* was placed in containers from which the light was excluded. It was so arranged that the parasites could leave the lighted tubes in which they were placed and enter the dark containers at will.

It was found that the parasites would enter the darkened containers in search of their prey without any hesitation, so that we may assume that their beneficial work will be carried on equally as well whether the corn is stored in the light or the dark.

Female parasites were frequently observed to feed on the body juices of the weevil grubs. After jabbing the grubs with their ovipositors they lap up the exuding juices.

PARTHENOGENESIS

Unfertilized females kept under observation laid fertile eggs which invariably developed into males. These females lived as long as the average fertilized ones or longer but laid only about half as many eggs.

NUMBER OF MALES AND FEMALES

Males are apparently more abundant than females. Of reared specimens 60 per cent were males.

LONGEVITY

The average female parasite in captivity lived for about 82 days. This period is considerably longer than the life of the male, which averaged 47 days.

One female parasite that was not allowed to deposit eggs but was fed on sugar and water lived from August 19 to December 4, 1920, a period of 107 days.

Parasites confined without food and without weevil grubs lived for only a few days; males so confined died by the end of the sixth day and females by the ninth.

COMPARISON OF LIFE-HISTORY STATISTICS WITH THOSE OF SITOPHILUS ORYZA

With an average length of life about half that of the rice weevil, this parasite lays fully as many eggs per day and completes its life cycle in a little less than half the time taken by the rice weevil.

A smaller percentage of females is produced by the parasite, however, and several eggs of the parasite may be wasted on one host grub, so that the rate of multiplication is below that of the rice weevil and a complete control is not obtained.

Table III gives a comparison of the life-history records of the parasite and the rice weevil.

TABLE III.—*Life-history statistics of Aplastomorpha vandinei and Sitophilus oryza compared* [1]

Insect.	Average length of life.	Average length of oviposition period.	Average number of eggs laid.	Average number of eggs laid daily.	Males emerging.	Females emerging.	Length of life cycle.
	Days.	*Days.*			*Per cent.*	*Per cent.*	*Days.*
S. oryza	111	93.9	380	4	48	52	35
A. vandinei	72	61	259	4	60	40	14

[1] Summer records.

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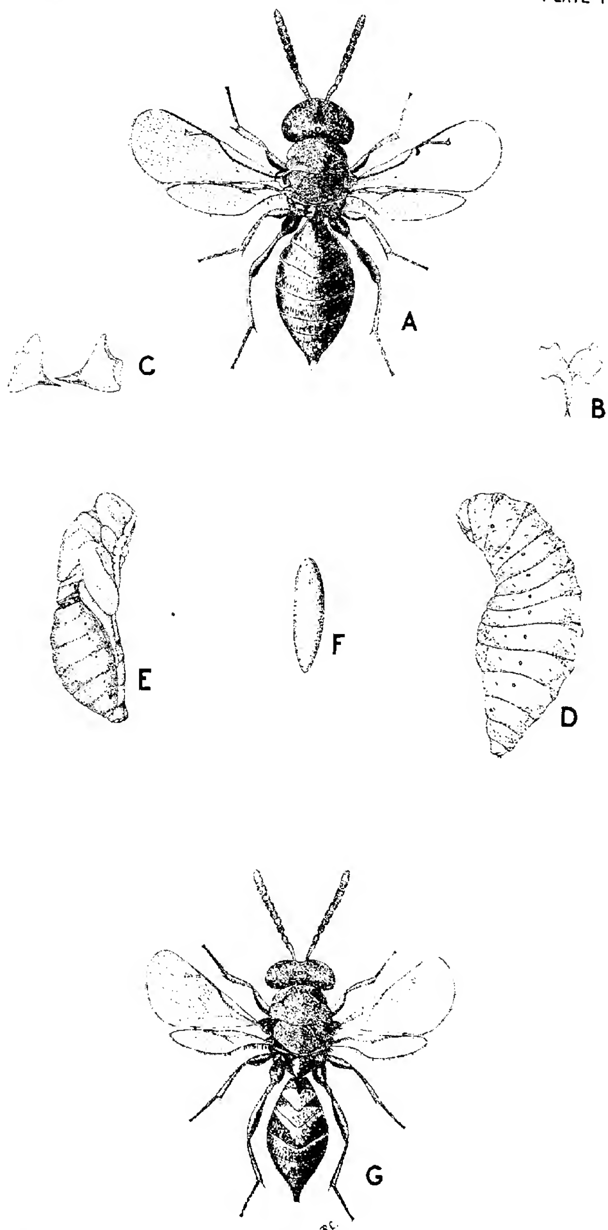
PLATE 1

Aplastomorpha vandinei

A.—Adult female.
B.—Mandible of first-stage larva.
C.—Mandible of mature larva.
D.—Mature larva.
E.—Pupa.
F.—Egg.
G.—Male adult.

Aplastomorpha vandinei Tucker

PLATE I



INHERITANCE IN SWINE[1]

3y EDWARD N. WENTWORTH,[2] *formerly Professor of Animal Breeding*, and JAY L. LUSH,[3] *formerly Fellow in Animal Breeding, Kansas State Agricultural College*

The genetic studies on swine described herein were begun on a rather :xtensive scale at the Kansas Agricultural Experiment Station in 1914, eached their maximum in the spring of 1917, at which time the senior uthor entered military service, and came to an end in the spring of 1918, when the junior author also assumed military duties.

With the exception of the second wild boar (Pl. 1, A), which is now the property of the Iowa State College, none of the swine used in this nvestigation are still living, and hence no further work can be done oward solving the unfinished problems, except by beginning once again vith a new group of individuals. Since there is no immediate prospect f this being done, it is thought best to publish the results secured, ogether with the most probable interpretation of them, although it is rankly admitted that the data do not furnish a definite solution of the roblems attacked and that in some cases they do little more than ndicate the general direction for future work.

Because there have been so few carefully controlled genetic experiments n swine it is felt that there is need to make this material available for urther study, even though many of the problems touched upon have ot been settled. This paper, therefore, is presented as a report of rogress.

PURPOSE AND PLAN OF THE EXPERIMENTS

The purpose of the experiments was to investigate the mode of inheri-ance of certain well-defined characters in swine, such as the shape of ice, set of ears, color, mammary pattern, growth factors, and the size f litter. On all these points, except size of litter, the principal work as done on the descendent generations from crosses of a registered erkshire boar by Tamworth and Duroc-Jersey sows. The problem of tter size was separately attacked through mating European wild boars the Schwarzwald type to registered Tamworth and Berkshire sows. he production of a large F_2 generation and the making of suitable ick crosses were planned, but only a single F_2 litter from the wild × amworth cross and none from the Wild × Berkshire or Berkshire × amworth cross had been secured when the work was discontinued. ence the majority of the data will concern the Berkshire × Duroc-rsey cross, from which a large F_2 generation and a considerable number back cross individuals were obtained.

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The following records were kept for each pig: (1) The number of pigs in the litter in which each individual was born; (2) descriptions of color, set of ears, and shape of face, the latter character being described from three different viewpoints—length of face, dish of face, and shape of forehead; (3) a diagram of the arrangement of the mammae; and (4) growth as indicated by monthly weights.

The total number and relationship of the pigs produced in each cross were as follows:

Wild × *Tamworth:* This cross comprised an F_1 generation of 38 pigs produced in 5 litters out of three Tamworth sows and by two wild boars; 1 back-cross litter of 8 pigs out of another Tamworth sow and by F_1 boar; and 1 F_2 litter of 4 pigs by the F_1 boar out of a litter sister.

Wild × *Berkshire:* In this cross an F_1 generation of 2 litters totaling 17 pigs was secured out of 1 registered Berkshire sow by the second wild boar.

Berkshire × *Tamworth:* This mating produced 1 litter of 10 pigs out of a Tamworth sow by a registered Berkshire boar.

Berkshire × *Duroc-Jersey:* The result of this cross was an F_1 generation of 29 pigs in 3 litters out of two registered Duroc-Jersey sows by two registered Berkshire boars, an F_2 generation of 151 pigs in 17 litters out of 8 F_1 sows by an F_1 boar, 1 F_3 litter of 11, 1 litter of 5 out of an F_2 sow by the F_1 boar, and a back-cross generation of 35 pigs in 3 litters out of the Duroc-Jersey sow that was the mother of all the F_1 animals reserved for breeding, by her F_1 son.

There is no *a priori* reason to assume that similar characters in two quite distinct breeds—for example, the erect ears of the Berkshire and of the wild hog—are due to identical factor complexes. However, since it is more convenient from the standpoint of treatment, each character will be discussed in the light of the data from all available crosses.

INHERITANCE OF LITTER SIZE

Inheritance of litter size was studied in two crosses (1) wild boar by Tamworth sows and (2) Berkshire by Duroc-Jersey.

It was primarily for the study of the inheritance of litter size that the wild hog was used in this experiment, its use having been suggested in a previous study by Wentworth and Aubel (*23*).[4] This type of wild hog normally produces about 4 pigs per litter, the Tamworth 11, and the other common breeds about 8 (*23*).

Obviously this character can be fully expressed only by sexually mature females, and since so many nongenetic factors (*23*) can operate to reduce the actual number of pigs in a given litter below that which the sow is potentially capable of producing, it will not be safe to draw far-reaching conclusions from the limited data available in this experiment.

Only one F_1 sow of the wild × Tamworth cross produced any litters, and she produced but one (Pl. 1, B). The fact that her litter consisted of but four, however, agrees with the results obtained by Simpson (*11*) and is very suggestive of the dominance of the wild litter size. It may be considered a good indication that litter size in swine, like fecundity in poultry (*8*), is dependent upon certain very definite factors which can be inherited in part through the male parent, since whatever factors

[4] Reference is made by number (italic) to "Literature cited," p. 581–582.

for wild litter size this sow possessed must have been received through her sire. The average size of litters produced by the four Tamworth sows in this experiment was not up to the breed average, being only 7.67 pigs. However, four of these six litters were gilt litters, produced before their dams had reached full maturity, which may partially account for their small average size. A direct influence of the sire on the size of the litter has been demonstrated only under the condition of his previous excessive sexual use (*6*), a condition which did not apply in this case.

The data in regard to litter size in all these crosses are summarized in Table I.

TABLE I.—*Size of litters*

Description of dams.	Total number of litters.	Number of gilt litters.	Extreme size of litters.	Mean size of litters.	Standard deviation of litter size.
Pure-bred Tamworth........	7	5	6 and 10	7.86	1.46± .26
F_1 wild×Tamworth.........	1	1	4	....	....
Pure-bred Berkshire.........	2	1	7 and 10	....	....
Pure-bred Duroc-Jersey.....	6	2	8 and 17	10.67	2.62± .51
F_1 Berkshire×Duroc-Jersey..	17	8	3 and 12	8.88	2.88± .33
F_2 Berkshire×Duroc-Jersey..	2	2	5 and 11	....	....

INHERITANCE OF SET OF EARS

Three of the breeds used in this investigation, the Berkshire, Tamworth, and wild, have erect, pointed ears of fine or moderately fine texture, and, since all of the pigs produced by intercrossing showed a very similar sort of ear, these crosses furnish no evidence as to the factors for inheritance of the ear shape. The Duroc-Jersey breed, however, is characterized by a quite different shape of ear; hence the Berkshire × Duroc-Jersey cross throws some light upon this question and will be discussed in more detail.

The Berkshire ear, in addition to being erect and pointed, is rather small and fine in proportion to the size and length of the body. It may droop somewhat with extreme age (*9*), but breaks forward at the head and not within the length of the ear.

The Duroc-Jersey ear, while not a direct opposite, offers a good contrast in several respects. It is of medium size, not so pointed, and the outer third of it breaks over sharply and droops downward. There is considerable variation in the amount of droop, but close inspection shows that these variations are rather closely correlated with variations in the general quality of the animal. Thus the large, extremely flabby ear, broken over more than breed ideals permit, is usually found on animals which show a general roughness and lack of quality. On the other hand, the extremely fine, almost erect type of ear is closely associated with a lighter limbed, neater quality type of hog. It is not known at present whether this correlation is genetic linkage or due simply to the mechanics of growth.

Both of the Duroc-Jersey sows and both of the Berkshire boars of the parental generation were typical of their breeds with respect to ear shape. The F_1 generation contained 29 pigs, and, with the exception of one boarr (Pl. 1, C, D) and one sow in the first litter, which was sired by a boar

not used subsequently, all of them, so far as could be observed, possessed perfectly erect ears, which, however, were slightly larger than on Berkshires of equal size.

Although the F_2 generation consisted of 151 individuals, only 42 had become mature enough to determine definitely the shape of ear when the work was discontinued. Of these only one (Pl. 1, F) showed a typical Duroc-Jersey ear; several showed shapes intermediate in various degrees, and the great majority showed typically erect Berkshire ears.

The Duroc-Jersey sow that was the dam of all the F_1 generation which was saved for breeding purposes was mated to an F_1 son from her first litter (Pl. 1, E). There resulted three litters totaling 35 pigs, most of which died young or were still young when the experiment was discontinued. Of the 9 which were 6 months old or more at that time, 4 had typical Duroc-Jersey ears, 1 was still undetermined, and 4 had ears completely or almost completely erect.

The following conclusions in regard to the inheritance of the set of ears are warranted by the data: The typical erect ear of the Berkshire is dominant by at least one and probably not more than three (F_2 ratio of 41 erect or intermediate to 1 of Duroc type) principal factors. There are probably a number of minor modifying factors for size and quality as well as for the amount of breaking over. Neither breed is homozygous throughout for all the factors concerned in the production of its own peculiar ear shape—that is, being pure-bred is not equivalent to being homozygous in this respect.

INHERITANCE OF SHAPE OF FACE

This character, like the shape of ear, does not complete its development until the animal is mature and is fairly constant within each of the breeds of swine.

Both the Tamworth and the wild hog are characterized by long, narrow, straight faces with almost no forehead prominence. The face of the wild hog is the more extreme in each of these respects, and the seven mature F_1 pigs all approached very closely to the face shape of their wild parent.

Among the common American breeds of hogs, the Berkshire represents the brachycephalic opposite to the wild hog in face shape, having a very short, extremely dished face and a forehead so broad and prominent as to give it a sort of pompadour appearance (Pl. 1, G). The facial angle, while varying somewhat like any other quantitative character, is fairly constant and approximates a right angle. Only two F_1 pigs from the wild × Berkshire cross matured, and both resembled the wild parent so closely that, except for color and wider foreheads, they would have been indistinguishable from the F_1's of the wild × Tamworth cross. One of them, however, also had a slightly shorter face than either his litter mate or the F_1's wild × Tamworth. Not enough data are available to indicate whether or not this was due to some variation in development.

Seven of the 10 pigs composing the F_1 generation of the Berkshire × Tamworth cross matured, and none of them could be distinguished from pure-bred Tamworths as far as face shape was concerned. This is especially surprising because Simpson (*16*), who crossed Yorkshires and Tamworths, reported that the Yorkshire face (which is quite similar in appearance in respect to dish of face to the Berkshire, though longer)

was neither dominant nor recessive, but that the faces of the resulting F_1 animals were intermediate between the two parental types. Simpson did not give the data for the F_2 generation, and none of the F_1 individuals in this experiment reproduced. Hence the number of factors responsible for the differences in face shape between these three breeds is not known. The data, however, indicate this much: Tamworth straight, long face is dominant over Berkshire short, dished face; Yorkshire dished face forms as intermediate F_1 with Tamworth long, straight face. Therefore, Yorkshire dished face and Berkshire short, dished face are apparently similar phenotypes produced by somewhat different factor complexes.

The face shape of the Duroc-Jersey is intermediate between those of the breeds just discussed, in that it is moderate in length and in dish, while the forehead and the hair which grows upon it usually slope smoothly upward and backward. In the F_1 generation of the Berkshire × Duroc-Jersey cross, the forehead in every case approaches closely that of the Berkshire type (Pl. 1, D). The face was strongly dished, but not to the degree of the Berkshire ideal, although it approached this ideal much more closely than the Duroc-Jersey type. A slight amount of variation was shown by two animals of the F_1 generation, but the dish was pronounced enough to be Berkshire in type (Pl. 1, H, I). The second of the two F_1 litters from which breeding animals were selected was much more typically Berkshire than the first. Both litters were out of the same dam, but they were sired by different boars. In every case the length of face was considerably less than the Duroc-Jersey and approached the Berkshire type. Here, too, there was some variation.

In the F_2 generation very much wider variation was observed. No practicable means of measuring any of the three elements of face shape was devised, but since they differed so distinctly, and there was so little chance of mistake, they were classified according to their resemblance to one or the other of the parental types. The face shape does not develop completely until the animal is mature, hence there are available only the data for the 42 F_2 animals which had reached maturity when the experiment was discontinued. These data are summarized in Table II.

TABLE II.—*Inheritance of facial characters in Berkshire-Duroc-Jersey cross*

Facial character.	Similar to Berkshire.	Intermediate.	Similar to Duroc-Jersey.
Forehead shape	37	2	3
Dish of face	17	9	16
Length of face	21	7	14

The widest variation was in the length of face, some being longer than the typical Duroc-Jersey and some shorter than the typical Berkshire. There was some evidence of correlation between length of face and dish of face since hogs both with extremely dished and extremely long faces did not occur. On the other hand, short-faced hogs occurred both with extremely straight and with extremely dished faces. This

may be interpreted to mean that such correlation as exists is developmental or anatomical rather than genetic (linkage).

The data from this cross seems to show that the Berkshire type of face is largely but not completely dominant over the Duroc-Jersey type. Probably several factors differing in importance and in degree of dominance are responsible for the anatomical differences that determine the face shape. Certain of these factors affect certain parts of the face much more than they do other parts. If reliable means of measurement had been developed for the different aspects of face shape, it might have been possible to distinguish enough separate phenotypes to postulate a factorial analysis. In the absence of such an analysis it would be impossible even to estimate the number of factors involved, beyond calling attention to the fact that the true number can not be so large as to make analysis impossible, else the parental types would not have been recovered among such limited numbers.

RÉSUMÉ OF WORK ON SWINE COLOR (24)

In spite of the rapidity of their multiplication and the diversity of characters which they offer, very little genetic research has been conducted with swine and that which is known or seems very probable in regard to the inheritance of their colors can be stated in a very few paragraphs (1, p. 476).

The agouti color common to so many mammals is found among swine in the European wild hogs. This color, which is fundamentally a pattern of bands of different shades of sandy, red, black, or brown pigment on each individual hair is a darker, more slaty shade in the adult wild hog than is usual among other mammals. It is also characterized by being much lighter along the underline than on the sides and back. Moreover, in the wild hog the young are born with longitudinal stripes instead of being self-colored.

The European wild hog (Pl. 2A) has been crossed with the domestic breeds of hogs by Simpson in Illinois (*11, 12, 13, 14, 15, 16*) and by Herrmann and by Henseler among others in Germany as stated by Frohlich (*4, p. 219, 220–221*) but as yet there has been no very large F_2 generation produced. In the F_1 generation the striping pattern of the young and the agouti pattern of the adult have been reported dominant over the self red of Tamworths and the black with six white points of the Berkshire and incompletely recessive to two patterns—that of a white belt on a colored background and that of self white (*4, 14, 15, 18*). Simpson's data from a cross with Tamworths indicate that this wild color differs from red by a single factor.

Each individual hair of domestic swine is either white, black, or some shade of red; but within these limitations there is a wide range of colors, not only in the scrubs and grades which constitute the average market run of hogs but also between the various pure breeds. Thus there are self red, self black, and self white breeds which breed true to pattern, besides a recently established breed which is a black and white roan or "blue." Also, although there are no pure breeds of these colors, ordinary unpedigreed hogs are common which are red-and-black, red-and-white, red-black-and-white, red-and-black roan in spots, or red-and-white roan in spots. Red may be replaced by various lighter tints, ordinarily known as sandy, in any of these combinations. There appears to be no record of true albinism in the pig. If it does occur it is cer-

tainly so rare that it plays little part in the ordinary colors. Self-white breeds are fairly numerous, there being at least five known in America and one, the Edelschwein, in Germany.

The Edelschwein self white has been found dominant to the striping and agouti pattern of the wild hog (*4*), and the Yorkshire and Chester White self whites have been reported dominant in the F_1 generations over one kind of Tamworth (*12*) and Duroc-Jersey self red, and over Hampshire, Berkshire, and Poland-China (*17*) black and has produced a red-and-white roan in the F_1 of a cross with some Tamworths and Duroc-Jerseys whose self red color seems to be genetically different from the more common red color of their breeds (*19, 20*). The black of Berkshire and Poland-China segregates in a 1 to 3 ratio in the F_2 generation, but the black pigs obtained as recessive carry more and larger white spots than the pure-bred Poland-China or Berkshire. Evidently there are some minor factors which modify the extent of this kind of black and which are inherited independently of the main factor for black or its allelomorph (*17*).

Another form of white which appears to be quite distinct, genetically, from self white and from the white spotting displayed by the Berkshire and Poland-China is the white belt, "sheet," or "list," as it is sometimes known to breeders, which is characteristic of the American Hampshire hog and, in a more extended form, of the German Hanoverian swine. This white pattern is dominant, although not always completely so, when crossed with all other colors upon which it has been tried, including even the striping and agouti pattern of the wild hog (*4*). The swine in this case were Hanoverian sows bred to a wild boar. The white belt of the Hanoverian is so extensive that usually the only parts which remain colored are the head and a part of the rump, while in the American Hampshire the white belt includes only the front legs and feet and a belt usually from 4 to 12 inches wide around the body. The two patterns are probably of the same kind genetically, except that the two breeds do not possess the same modifying factors for the width of the belt. Even in pure Hampshires, the inheritance of the belt can not be determined by less than two factors (*19, 20*). Simpson has succeeded in producing belted red hogs by crossing the Hampshire and Tamworth and extracting reds from later generations (*12*).

A second kind of white pattern is that possessed by the Poland-China and Berkshire breeds. These are black except for white feet, a white splash in the face, and white on the end of the tail. These six white points constitute the standard color for both breeds, but quite frequently there are one or more white splashes elsewhere on the body, and occasionally an animal appears which is not pure white but a sandy color in one or more of these "white points." The complete pattern is manifestly not dependent on a few factors, since it breaks up so readily into black spotting on a white, sandy, or red ground color in crosses and is so difficult to obtain in typical form in the F_2 generation.

A possible third kind of white pattern is found in the breed known as the Spotted Poland-China. White and black, in approximately equal amounts irregularly scattered over the body, constitutes the standard color of this breed. Wright suggests (*24*) that it is doubtful whether this is really a different kind of white pattern from that of the Berkshire and Poland-China, since both of these breeds at a not very distant time were sandy white or reddish brown with large black spots. Furthermore when they are crossed their present color readily breaks up into a form

of black spotting which appears similar to that of the spotted Poland-China. Wright comes to the conclusion that the Berkshire and Poland-Chinas are genetically black-and-sandy spotted hogs in which, by selection of minor factors, sandy has been diluted to white and black has been extended to cover all the body except the six white points. Such evidence as the writers have with regard to the white points casts no light on this interpretation, but the idea seems correct for the black color.

Aside from the fact that the roans occur regularly as a part of the progeny resulting from back-crossing Yorkshire×Tamworth F_1 hogs to pure Tamworths (*12*) and in the F_1 generation of self white breeds crossed with Hampshires (*5*) nothing definite is known about the inheritance of the roaning patern. The Sapphire, the only breed making a feature of this pattern is of such recent origin that it does not yet breed completely true and hence is not suitable for analyzing this character (*7*). Several types of roans have appeared in the course of the experiments to be reported upon here but not in sufficient numbers nor with sufficient regularity to permit their analysis. As a general rule, roans appear to be either self colored or ordinarily spotted at birth, but at about weaning time hairs of another color begin to appear and soon produce the roan effect, either as a self roan or a spotted roan.

Two self black breeds are known in England but are not common in America and their color behavior in crossing is not known, although the black of the Large Black breed has been reported dominant to Tamworth red.

Hampshire black is certainly distinct, genetically, from Berkshire or Poland-China black, but whether it is equivalent to self black plus the white belt or is a third distinct kind of black is not known (*5*).

Self red breeds vary in the intensity of their pigment rather widely from a light yellow or sandy to a deep brownish red which almost approaches black. That these differences in intensity are hereditary seems probable, but no definite investigation of this point has yet been made except to find that there are two distinct kinds of red with respect to the way they react towards self white (*19, 20*). Self red is recessive to self white, to the wild pattern, and to Hampshire black with white belt. Self red is the only type of red which is characteristic of any pure breed, but grade and scrub hogs are common which are spotted red with black or white or roan in various combinations.

This general survey of the heredity of swine color, so far as it is known, may be briefly summarized as follows:

Self white. Dominant over all other colors. Probably dependent on a single factor.

White belt. Dominant over all other colors but must depend upon more than one factor.

Immature striping and adult agouti of the wild hog. Dominant over all other colors except self white and the white belt. Dominance is not complete.

Roaning. Appears frequently among descendants of self whites by self reds, or of self whites by belted blacks.

Self black. Dominant to self red.

Black spotting on a lighter ground color. The most frequently occurring type of black in ordinary market hogs and therefore probably dependent upon a few relatively simple dominant factors.

Self Red. There are two kinds of red, genetically distinct in their behavior toward self white. Possibly the factor or factors which affect the white may be distinct from the factors for red.

COLOR DATA FROM THIS INVESTIGATION

WILD BY TAMWORTH CROSS

The color of the Tamworth is a uniform cherry red which varies somewhat in intensity within the breed but not so widely as the red of the other common red breed, the Duroc-Jersey. The color of the wild hog has already been described. All of the 38 F_1 pigs produced in this experiment were born with distinct longitudinal stripes about 1 cm. in width, composed alternately of rather light red hairs and very dark brown hairs. These stripes extended all over the backs and sides, but the bellies were a uniform light red (Pl. 1, B). Such variation as may have existed among the F_1 individuals in the regularity of striping or in the contrast between light and dark stripes was too slight to admit of description or of measurement.

All four F_2 pigs were striped in the same way except that the stripes were not quite so regularly continuous throughout the entire length of the pigs' bodies as was the case with the F_1's. These four pigs showed, however, three different belly colors, as follows: One with a uniform grayish white belly on which the hairs were apparently white, two with uniform, light reddish bellies like the F_1s and one with a similar light reddish belly on which were scattered some large black spots both in hair and skin.

The back-cross litter resulting from mating a Tamworth sow and an F_1 boar included five striped and three nonstriped pigs. Of the five striped ones, two were like the F_1's and three were faintly striped but possessed small black spots on their bodies. Of the nonstriped pigs, one was self red and the other two were red with small black spots.

Obviously this suggests single independent factors for black spotting and for the striping pattern. It also suggests that the black spotting factor may diminish the intensity of the striping pattern, because all three of the back-cross pigs which were faintly striped possessed black spots. Since none of these pigs matured, it was impossible to apply the breeding test to this latter idea, but the existence of an F_2 pig which was both intensely striped and possessed black spots makes it seem more probably that the association of faintness of striping and black spotting in the back-cross litter was due either to chance or to linkage. The factors for striping pattern and for intensity of that pattern must both come from the wild hog, while the factor for black spotting probably comes from the Tamworth, since if it comes from the wild hog the latter must also possess an inhibiting factor which prevents the spots from showing in either the F_1 or the pure wild. The occurrence of the gray-bellied pig in the F_2 generation and the absence of this type from the back-cross litter suggests that the Tamworth may possess a partially dominant factor for deep red and the F_2 pig may be explained as the segregation of the homozygous recessive to that factor. This last suggestion is supported by the fact that the wild × Berkshire F_1 pigs, to be discussed later, showed this lighter color and by the fact that the adult wild × Tamworth F_1's were a more reddish and less slaty color than the pure wild boars. This is the only actual evidence secured supporting this interpretation. No evidence was secured as to why the pure-bred Tamworth, which evidently carries a fundamental factor for black spotting, is prevented from showing that black in its own body.

WILD BY BERKSHIRE CROSS

All the 17 F_1 pigs which composed this cross were fairly uniform and striped, and, except in two particulars, closely resembled the wild×Tamworth F_1 pigs. They had much less of a reddish tinge throughout and showed a number of large black spots along the underline and on the face, ears, and legs. The lack of the reddish tinge made the dark stripes appear a blacker brown and the light stripes and underline a very light gray. The black spotting was different in appearance from that in the wild×Tamworth F_2 and back-cross pigs in two particulars; first, the spots were larger, usually being from 1 to 1¾ inches in diameter on the new-born pigs, while those on the pigs of the Tamworth cross were rarely more than ¾ inch in diameter; secondly the spots on the latter tended to be restricted to the rear half of the animal, being especially frequent on the thighs and on the underline behind the umbilicus, while the larger spots on the pigs of the Berkshire cross appeared with almost equal frequence on all parts of the underline and lower sides, although perhaps a little oftener on the lower law, the side of the face, and the lower half of the ears.

From these data it seems that the Berkshire lacks the factors determining the deeper red color which the Tamworth possesses and has either a different fundamental factor or quite different modifying factors for black spotting because this spotting shows up at once in the F_1 generation of the wild×Berkshire cross and because it is different in appearance.

BERKSHIRE BY TAMWORTH CROSS

The 10 F_1 pigs composing the only litter of this cross consisted of 8 which were red with one or more small black spots and 2 which were self-red. The red varied in intensity from a reddish gray to a dark reddish brown. Four of the 8 had many small black spots, 3 had only a few, and 1 had but a single black spot on the side of her nose. Therefore, it seems possible that there was no genetic difference between the 2 self-red pigs and the 8 with black spots in regard to the fundamental factor for black spotting, the variation being due to differences in development or differences in modifying factors for restriction of black. These pigs had, on the average, fewer and smaller spots and were of a deeper red body color than the F_1 pigs from the Berkshire by Duroc-Jersey cross to be discussed next.

BERKSHIRE BY DUROC-JERSEY CROSS

The Duroc-Jersey, as a breed, shows considerable variation in the shade of red, but the sows of the parental generation of this cross were of medium color, neither very light nor very dark. The one whose son and daughters were used to produce the F_2 generation and that was herself the dam of all the back-crossed litters was the desired cherry red. She was a good specimen of Duroc-Jersey brood sow and typical of the breed in every way except, perhaps, that she was chuffier than the breeder desires and her face was a little more dished than the average of the breed.

The 29 pigs composing the F_1 generation all possessed small black spots scattered irregularly over a yellowish red (in most cases, sandy) body color. The black spots appeared a little more numerous on the

underline and rear parts, but this was not uniformly true. The spots were all small, very few of them being over 1 square inch in extent on the new-born pig and most of them being not more than one-third as much. The red body color varied in intensity, 4 of them being described as sandy red, 2 as red with white bellies, and 1 as red with a sandy belly. Uniformly as they matured the red became lighter and, although they did not all come to the same shade of red then, it would be more accurate to describe them, when mature, as sandy hogs with numerous small black spots. Where not otherwise stated in this discussion, the color of a pig is understood to mean the color at time of birth because red and sandy were then most distinct.

Eight sows and one boar (Pl. 2, B) all of which were described as black-and-red at birth, were selected from the F_1 generation for breeding purposes and from them were produced 151 pigs (Pl. 1, J), including three which ceased development at such an early prenatal stage that their true color could not be distinguished with certainty. When classified as to the color of hair, without regard to the patterns in which these colors were arranged, the 148 F_2 pigs and the 35 back-cross pigs produced by breeding the F_1 boar back to his own dam, gave the results shown in Table III.

TABLE III.—*Color of 148 F_2 pigs of the Berkshire-Duroc-Jersey-Cross*

Color.	Number in F_2.	Number in the back-cross.
Black-and-red	47	19
Black-and-white	27	....
Self red	14	14
Black-and-sandy	11	....
Black-red-and-white	10	1
Black-red-and-sandy	10	....
Sandy	7	....
Sandy-and-white	7	....
Black-sandy-and-white	6	....
Red-and-sandy	3	1
Red-and-white	3	....
White	3	....
Total	148	35

A classification according to the presence or absence of each color separately gives the results presented in Table IV.

TABLE IV.—*Color of pigs in F_2 of the Berkshire-Duroc-Jersey-Cross, classified as to each color separately*

Color.	Number in F_2.	Ratio.	Number in the back-cross.	Ratio.
Showing black	111	3:1 (exact)	20	1:1 (approx.)
Not showing black	37		15	
Showing red	87	9:7 (approx.)	35	....
Not showing red	61		....	
Showing sandy	44	1:2.3	1	....
Not showing sandy	104		34	
Showing white	56	1:1.6	1	....
Not showing white	92		34	

The most obvious color, and the one about which there is the least chance for mistake,[5] is the black, and since it gives an exact 3 to 1 ratio in F_2 generation with such a relatively large number of individuals and such a close approximation to a 1 to 1 ratio in the back-cross, it is rather clearly evident that the presence of black color, irrespective of whatever pattern it may assume, is due in this cross to a single dominant Mendelian factor received from the Berkshire.

In a few cases it was rather difficult to distinguish accurately between red and sandy and between sandy and white, but, on the whole (Pl. 2, C), these colors segregated distinctly, and in the case of certain combinations such as black-and-red or black-and-white there was never any question. This distinctness of color was most marked when the pigs were young because some became darker as they grew older while others became lighter. There were different shades of sandy and different shades of red, but these are not considered in detail in this discussion.

The ratio of the pigs showing red to the number not showing any red is very suggestive of a 9 to 7 ratio in the F_2 generation, and the ratio in the back-cross seems to prove definitely that at least one dominant factor whose function is to produce red pigment, rather than sandy or white, is possessed by the Duroc-Jersey breed.

From the ratios of sandy to nonsandy and white to nonwhite we get definite proof only that sandy is not a heterozygous red, since the sandy pigs are so much less numerous than the reds, and that white is not due simply to an absence of factors for black and red and sandy.

The independence in inheritance of the black color and distribution of spotting with black make it possible to regard red or sandy (that is, the possession of red pigment) as the fundamental ground color of the hog. In order to get at the relation of red, sandy, and white, therefore, it may be permissible to examine them, disregarding whatever black may be shown on the bodies of the animals. If the black color is omitted from Table III, the result shown in Table V is obtained.

TABLE V.—*Distribution of red, white, and sandy colors in Berkshire-Duroc-Jersey F_2 generation*

Color.	Number in F_2.	Number in the back-cross.
Red	61	33
White	30	......
Sandy	18	......
Red-and-sandy	13	1
Red-and-white	13	1
Sandy-and-white	13	......
Total	148	35

Let us first consider the relation of red and sandy. Omitting white on the theory that it is due to superimposed spotting factors, there are 87 reds (61 red, 13 red-and-white, and 13 red-and-sandy) to 31 sandy (18 sandy and 13 sandy-and-white). This misses a true 3 to 1 ratio (88½ to 29½) very slightly and suggests a single intensity factor difference

[5] Three of the pigs of the back-cross were classified as black-and-red although they possessed only one small black spot each, and in one of these cases the black was in the skin only and not in the hair growing on it. If either of these three cases has been placed in the wrong genetic classification, the correct ratio in the back-cross would still more closely approximate 1 to 1.

for red and sandy. It may be protested that the red and sandy pigs could as logically be included under sandy, but the writers have not accepted this idea because in each case where red and sandy occurred on the same hog, the sandy markings were so distributed as to indicate a failure of the intensity factor for red, just suggested, to extend to the extremities of the animal, in much the same way that the factor for restriction of black pigment in the bay horses varies centrifugally. Sandies do not appear in the back-cross to permit the testing of this hypothesis.

The data with regard to white spotting are less suggestive. Omitting the three individuals that were self white (Tables IV and V), there were 53 with white markings in the F_2 generation and 92 without. This indicates three possibilities: First, that white markings are not dominant unless dependent on the interaction of three or more factors; second, that white markings are recessive; or, third, that they are dependent on several factors, some of which are dominant and others recessive. The data are not complete enough to test any of these hypotheses. However, if the first hypothesis is correct, three or four factors are indicated. If the interaction of three factors is necessary, the expectation for 145 F_2's would be 61.2 with white markings to 83.8 without. If the interaction of four is necessary the expectation would be 45.9 with white markings to 99.1 without. The actual ratio, 53 to 92, is about half way between the two. There is no simple recessive condition that will produce this ratio, nor is there any combination of recessive conditions probable that would give the actual results. Hence the third hypothesis is most probable. In fact, three distinctly different types of white markings are readily recognizable in addition to the red-and-sandy "bicolor." The first is roaning; the second is the broken splotching of white, probably related genetically to the Berkshire "six white points," and the third is the light belly marking. The indications are that sandy may be substituted for white in many cases.

Roaning appeared in three forms. Two animals were red-and-white roan with black spots, one was sandy-and-white roan with black spots, and one was white with black-and-red roan spots. The roaning was not evident until after weaning time, and hence many of the pigs which died young also may have possessed this character. No attempt is made to analyze or explain the roaning condition.

The second form of bicolor was the irregular spotting of red or deep sandy upon a white or light sandy ground color. It always occurred in the presence of black and was readily distinguished from other colors by the fact that the red spots were always most numerous on the face, back of the head, and front part of the back. In nearly every case of this type one or both upper eyelids and eyelashes were quite red. The distinctness of this type of red spotting was not suggested until late in the investigation and consequently exact numbers are not available; but since it always occurred in the presence of black and did not appear among the back-cross individuals the suggestion is very plausible that this red spotting is genetically identical with the red or sandy splash which occasionally appears on the pure-bred Berkshire. The fact that the sandy color appears on the white splash on the face more commonly than on the feet or tail of pure-bred Berkshires supports this suggestion, and the fact that a pure-bred Berkshire practically never has all six "white points" showing sandy is another argument in favor of it. This

spotting may not be apparent in all individuals carrying it because the spots may all happen to be covered epistatically with black pigment.

The third form of bicolor appears as a red with a sandy or white belly (Pl. 2, D), or a sandy with a white belly. Black may or may not be present, but the occurrence, or at least the observance, of this character is more frequent with the nonblacks. Both the bicolor reds occurring in the back-cross and all three of the bicolor reds in the F_1 generation were of this type. The line of demarcation between the two colors may be sharp or they may blend into each other. This type of color—that is, a lighter underline than sides or back—is common to most mammals as an integral part of the agouti pattern. It is striking in the young of the wild hog, which also exhibit both (if they are genetically separate) the agouti and the striping patterns. For reasons to be discussed later it is considered probable that the three characters of lighter underline, striping pattern, and adult agouti pattern are due either to identical or to closely similar factor complexes.

A most interesting peculiarity of the F_2 Berkshire-Duroc generation was that two of its individuals, which were otherwise a light sandy with white bellies, distinctly showed longitudinal stripes of a pattern similar to that of the young of the wild hog, but not nearly so intensely colored and hence not so contrasted. They were also not so permanent and disappeared within a very few weeks after birth. In the founding of the Sapphire breed, according to McLean (7), the striping pattern appeared several times, and the stripes in some individuals persisted throughout life. Simpson also reports (13, 11) the appearance of striped individuals in crossing several breeds, of which he mentions specifically a Tamworth by Yorkshire cross and a Berkshire by Poland-China cross. This striping pattern as seen in a hog of unknown ancestry found on a farm in Wisconsin is shown in Plate 2, E. Considering everything, this phenomenon of the striping pattern in pigs is quite comparable with the appearance of barring on pigeon wings when distinct breeds are crossed and with the dorsal and shoulder stripes sometimes seen on mongrel dun-colored horses, phenomena which have been discussed since Darwin's time.

There was one litter of pigs of the F_3 generation which threw some light on this question in an unexpected manner. A white F_2 sow (which had, however, patches of black pigment in her skin although the hair growing out of these patches was quite white) when mated with an F_2 boar (Pl. 2, F) which was red with a sandy belly produced the litter of 11 pigs shown in Plate 2, G. All were some shade of sandy or red, but also all had light bellies and all were striped even more distinctly than either of the F_2 Berkshire by Duroc striped pigs. None of them showed any trace of a skin spot of black pigment. Four were classified as dark red, 5 were light red, 1 was a sandy red, and 1 was a very light sandy, almost white. The red pigment on the dark red ones was more of a brown than on any F_2 individual and their stripes were very distinct. Neither parent was observed to be striped and only the sire exhibited the lighter belly, but it is conceivable that the dam possessed the factors for both (if they are two) patterns, but did not have enough pigment in her hair for the expression of either. Although a complete factorial analysis has not yet been achieved, this much is certain; the striping pattern and the lighter underline are closely related and the factors which produce them are relatively few and simple in the mode of their expression, else such a combination would not have appeared uniformly in such a

large litter nor separately in two other litters. There is probably nothing more mysterious about this case of "reversion" than there is about any other case of the complementary action of factors. The indicated fewness of the factors affords hope for their future analysis.

The first step in investigating the nature of the genetic differences between the red, sandy, and white pigments was to discover whether the presence of the black pigment affects the other three in any way. To do this, Table III was rearranged with the black carriers separated from the nonblacks. The result, together with the numbers to be expected on the theory that the blacks and nonblacks of each combination of red, sandy, or white should give a 3 to 1 ratio, is shown in Table VI.

TABLE VI.—*The relation of black pigment to the distribution of red, sandy, and white.*

Color.	Number which also show black.	Number not showing black.	Expected blacks.	Expected nonblacks.
Red	47	14	45.75	15.25
Red-and-sandy	10	3	9.75	3.25
Red-and-white	10	3	9.75	3.25
Sandy	11	7	13.5	4.5
Sandy-and-white	6	7	9.75	3.25
White	27	3	22.5	7.5

It will be seen at a glance that the proportion of animals which had any red on them but lacked black color to those of the same color with the addition of black was almost exactly 1 to 3, or, in other words, the inheritance of the black is independent of the inheritance of the red (disregarding the pattern of the color). The numbers of the black and nonblack sandy and sandy-and-white pigs were too small for certainty, but they were far enough from the expectations to indicate a disturbing influence at work here. The number of whites was large enough and the actual ratio was aberrant enough to permit the positive assertion that the black-and-white pigs were not simply white pigs to which black had been added. There was also a difference in the appearance of the white hairs in self whites and in black-and-whites. No microscopical examinations were made, but the white of the black-and-white pigs was a more lustrous white at birth and tended much more to remain quite white all through life than that of the pigs born self white. One of the latter developed a sandy tinge at maturity, and two pigs born a very light sandy became quite white at maturity, indicating that the very dilute sandies approach very closely to the whites. The whites are certainly not albinos, for their eyes are always pigmented, and one of them had large patches of black pigment in her skin, although the hair growing out of those patches was quite white.

The second step in investigating the nature of the genetic differences between the red, sandy, and white pigments was to rearrange Table IV, classifying the bicolor reds according to the darker pigment which they showed. That the greater intensity of red represents the true pigment condition if there were no interference from bicolor factors is indicated by the fact that it was always the greater intensity of red which appeared as spots upon the lesser intensity. This classification is given in Table VII.

TABLE VII.—*The relative frequency of reds, sandies, and whites in the F_2 generation*

Color.	Blacks.	Expected blacks.	Non-blacks.	Expected noublacks.
Red	67	62.4375	20	20.8125
Sandy	17	20.8125	14	13.875
White	27	27.75	3	2.3125

The results for the nonblacks are as good as could be desired for a 9 to 6 to 1 ratio, and at once suggest that where black is absent the color is determined by two dominant factors, each of which produce by itself a sandy color and which together produce red, while the absence of both results in white. The results for the black-carrying pigs are a moderately close approximation to a 9 to 3 to 4 ratio. They suggest that there is one factor for sandy and a separately inherited factor which intensifies sandy to red, as indicated in the discussion of Table V.

In order to reconcile the two viewpoints indicated, it is possible that the same conditions apply among blacks and nonblacks, except that there is also a dilution factor linked to black which can inhibit one of the factors causing sandy pigment, in the absence of the other, thus producing white. The factorial representation dependent on this suggestion follows:

S_1=first factor producing sandy pigment.	s_1=its absence.
S_2=second factor producing sandy pigment.	s_2=its absence.
D=dilution factor linked to black inhibiting S_2.	d=its absence.
B=factor causing black pigment.	b=its absence.

On this hypothesis the factorial representation of the zygotes of the two breeds would be as follows:

Berkshire $=\overline{BD}\ \overline{BD}\ s_1\ s_1\ s_2\ s_2$.
Duroc-Jersey $=\overline{bd}\ \overline{bd}\ S_1\ S_1\ S_2\ S_2$.
F_1 $=\overline{BD}\ \overline{bd}\ S_1\ s_1\ S_2\ s_2$.

The F_2 generation carrying black would then be:

Blacks and reds:
$1\ \overline{BD}\ \overline{BD}\ S_1\ S_1\ S_2\ S_2$. $2\ \overline{BD}\ \overline{bd}\ S_1\ S_1\ S_2\ S_2$.
$2\ \overline{BD}\ \overline{BD}\ S_1\ S_1\ S_2\ s_2$. $4\ \overline{BD}\ \overline{bd}\ S_1\ S_1\ S_2\ S_2$.
$2\ \overline{BD}\ \overline{BD}\ S_1\ s_1\ S_2\ S_2$. $4\ \overline{BD}\ \overline{bd}\ S_1\ s_1\ S_2\ S_2$.
$4\ \overline{BD}\ \overline{BD}\ S_1\ s_1\ S_2\ s_2$. $8\ \overline{BD}\ \overline{bd}\ S_1\ s_1\ S_2\ s_2$.

Blacks and sandies:
$1\ \overline{BD}\ \overline{BD}\ S_1\ S_1\ s_2\ s_2$ $2\ \overline{BD}\ \overline{bd}\ S_1\ S_1\ s_2\ s_2$
$2\ \overline{BD}\ \overline{BD}\ S_1\ s_1\ s_2\ s_2$ $4\ \overline{BD}\ \overline{bd}\ S_1\ s_1\ s_2\ s_2$

Blacks and whites:
$1\ \overline{BD}\ \overline{BD}\ s_1\ s_1\ S_2\ S_2$ $2\ \overline{BD}\ \overline{bd}\ s_1\ s_1\ S_2\ S_2$
$2\ \overline{BD}\ \overline{BD}\ s_1\ s_1\ S_2\ s_2$ $4\ \overline{BD}\ \overline{bd}\ s_1\ s_1\ S_2\ s_2$
$1\ \overline{BD}\ \overline{BD}\ s_1\ s_1\ s_2\ s_2$ $2\ \overline{BD}\ \overline{bd}\ s_1\ s_1\ s_2\ s_2$

The F_2 generation lacking black would then be:

Reds:
$1\ \overline{bd}\ \overline{bd}\ S_1\ S_1\ S_2\ S_2$
$2\ \overline{bd}\ \overline{bd}\ S_1\ S_1\ S_2\ s_2$
$2\ \overline{bd}\ \overline{bd}\ S_1\ s_1\ S_2\ S_2$
$4\ \overline{bd}\ \overline{bd}\ S_1\ s_1\ S_2\ s_2$

Sandies:
$1\ \overline{bd}\ \overline{bd}\ S_1\ S_1\ s_2\ s_2$
$2\ \overline{bd}\ \overline{bd}\ S_1\ s_1\ s_2\ s_2$
$1\ \overline{bd}\ \overline{bd}\ s_1\ s_1\ S_2\ S_2$
$2\ \overline{bd}\ \overline{bd}\ s_1\ s_1\ S_2\ s_2$

Whites: $1\ \overline{bd}\ \overline{bd}\ s_1\ s_1\ s_2\ s_2$

The expectations on this basis are shown in Table VII. Aside from the superficial agreement between the expectations quoted and the actual numbers, several additional facts supporting the hypothesis may be mentioned. In both the blacks and nonblacks the reds constitute nine-sixteenths of the total number, and therefore red pigment depends upon the complementary action of two factors. From the results of the back-cross both factors must be present in the Duroc-Jersey. Also the black-and-whites constitute one-fourth of the black-carrying pigs, and therefore differ from the F_1's by one important factor. Although the numbers are small, there is one bit of additional evidence in favor of the last conclusion. A black-and-white F_2 sow mated back to her sire produced five pigs. One was all white, two were black-and-white, and two were black-and-red. One of the latter had a sandy belly. On the theory that the white of the dam differed by only one factor from the red of F_1 the expectation was that half the pigs would show white, while upon a theory that white depends upon the absence of two factors, whether black was present or not, only one-fourth of them would have shown white.

One other point in regard to color pattern remains to be discussed which is of particular interest from the standpoint of determining the color factors responsible for the large amount of black on the pure-bred Berkshire. That point is the size and extent of the black spots on the pigs showing black. In every one of the 47 black-and-red pigs the black was present in small spots and covered relatively little of the area of the pig's body (Pl. 2, H). What little unevenness of distribution of the black spots occurred in this group of animals consisted in the spots being a little more frequent along the underline and on the posterior parts of the animals. In some of the sandy-and-black animals the spots were similar to those on the black-and-reds; in others the spots were much larger and more numerous. In most of the black-red-and-whites, black-sandy-and-whites, and black-red-and-sandies the black was relatively abundant, often covering more than half of the animal. Every one of the black-and-whites (Fig. 19) possessed more black than the average of the black-and-reds, and the average amount of black on the black-and-whites exceeded that of any other group, being probably a little more than half the body surface. A large amount of variation was shown among the black-and-whites, for they varied from as little as about one-tenth black up to almost the Berkshire amount. Since no pig with absolutely perfect Berkshire markings was produced (Pl. 2, J) the evidence indicates that the Berkshire pattern is composed of the fundamental factor for black spotting, a factor for restricting or diluting the sandy pigment, and at least two or three, probably more, independent factors which operate to extend the black. The small amount of black in the F_1 generation, both of the Berkshire by Tamworth cross and of the Berkshire by Duroc-Jersey cross, and the fact that all red-and-blacks of the F_2 generation carry a similarly small amount of black are strong evidence either that there is a factor which strongly restricts black and is linked with red, or that, as Wright suggests (*24*), the competition between the two processes of pigment formation precludes the possibility of a pig possessing both an intense red and a large amount of black. The large average amount of black and the wide variation in the pattern of the black-and-white pigs indicate that the extent of the black spotting is influenced by many extension and restriction factors, even when free from the influence of the red.

CONCLUSIONS WITH RESPECT TO COLOR

The wild hog carries a single factor for the immature striping (adult agouti) pattern and a separate factor which intensifies that pattern.

There is an hypostatic factor for black spotting carried by the wild or by the Tamworth, manifested in the cross-bred specimens from either breed.

The Tamworth carries a more effective factor complex for the restriction of black than does the Duroc-Jersey. The factor for wild color and pattern is not completely dominant to the factor or factors for Tamworth red.

The Berkshire carries a single factor for black spotting and numerous independent factors for the extension of that black. Probably the Berkshire also carries dilution or restriction factors for sandy pigment and some individuals carry a factor for sandy spotting.

The Duroc-Jersey carries two factors which together produce the red color of that breed. Cumulatively one acts as an intensifier to the other. This breed lacks the factor for black spotting but possesses, probably linked to one of the factors for red, at least one factor and probably more for the restriction of black.

The relations between the various shades of red, between the various shades of sandy, and between sandy and white when black is absent are not clear.

There are three somatically distinct types of red "spotting." One is roaning, a second is an irregular spotting of red on a lighter ground color, and the third is a lighter color of belly than of sides or back. This last form is closely related to the agouti and striping patterns.

Striping in domestic swine is a rather simple case of "reversion."

It is not expected that all of these conclusions will stand in every detail the test of further research. They do, however, afford hypotheses of color inheritance in the breeds of swine investigated and offer a working basis for further research by which they may be extended and revised.[6]

DIFFERENCES IN GROWTH

The economic importance of some definite knowledge in regard to factors for rapid growth may be surmised from the extent of the practice of cross-breeding hogs in order to secure the larger size and greater vigor of the F_1 hogs for market purposes.

Only the Berkshire × Duroc-Jersey cross was extensive enough to furnish significant data upon this point. Since they were not all born at the same season nor even in the same year, it is obvious that they were not exposed to identical weather conditions nor fed rations as identical as would be desirable in a nutrition experiment. However, it was the aim always to feed the best possible ration to produce the maximum practicable gains and it is believed that the data are, on the whole, reasonably comparable.

The data for the variability of the F_1 and F_2 generations are presented separately in figure 1, where the curves show the coefficients of variability for the monthly weights for both generations from birth up to the age of 14 months.

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[6] Since this was written a report (3) of the results of a cross between mule-foot and Duroc-Jersey swine has been published which agrees closely with two of the main conclusions reached in this experiment, namely, that the presence of black as contrasted to its absence is dependent upon a single factor and that the different shades of red are due to the interaction of a few independent factors which do not affect black. The black of the mule-foot swine behaved like that of the American Hampshire as far as it has yet been analyzed, but whether the two are genetically identical is still undetermined.

Table VIII presents the data from which these curves were constructed. The decrease in the number of individuals indicated in the earlier months was due both to deaths and to the fact that many of the animals were still young when the experiment was discontinued. After the eighth month the decrease was due mostly to sales. This, by culling out the larger individuals, may have been responsible for a certain amount of decrease in variability of the F_2 generation but was without effect upon the curve of the F_1 generation for there were no sales from it and the deaths seemed to be normally distributed among the large and small animals.

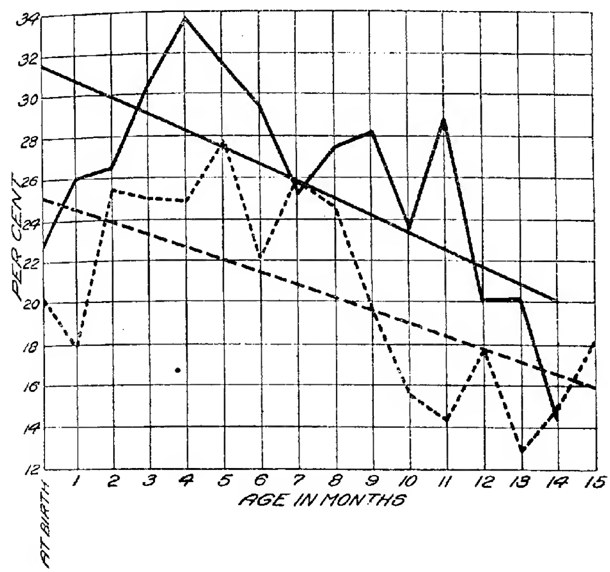

FIG. 1.—Coefficients of variability for the monthly weights of the F_1 and F_2 generations of the Berkshire X Duroc-Jersey cross. The solid line represents curve and fitted straight line for the F_2 generation, the dotted line the curve for the F_1 generation, and the broken line the fitted straight line for the F_1 generation

TABLE VIII.—*Variability of the F_1 and F_2 generations.*

Age.	F_1 Number of individuals.	F_1 Mean weight.	F_1 Standard deviation.	F_1 Coefficient of variability.	F_2 Number of individuals.	F_2 Mean weight.	F_2 Standard deviation.	F_2 Coefficient of variability.
		Pounds.				*Pounds.*		
Birth	13	2.46	0.498	20.3	136	2.4	0.547	22.77
One month	20	13.2	2.36	17.88	102	10.07	2.62	26.02
Two months	20	25.65	6.55	25.54	83	18.89	5.c	26.47
Three months	19	35.05	8.79	25.08	56	32.88	10.04	30.54
Four months	18	47.72	11.89	24.92	55	46.29	15.52	33.53
Five months	17	61.94	17.14	27.67	51	64.71	20.31	31.39

TABLE VIII.—*Variability of the F_1 and F_2 generations*—Continued.

Age.	F_1				F_2			
	Number of individuals.	Mean weight.	Standard deviation.	Coefficient of variability.	Number of individuals.	Mean weight.	Standard deviation.	Coefficient of variability.
		Pounds.				*Pounds.*		
Six months	15	86.0	19.01	22.1	48	87.54	25.76	29.43
Seven months	15	115.6	30.04	25.99	43	113.0	28.58	25.29
Eight months	13	135.61	33.18	24.47	43	130.46	36.0	27.59
Nine months	13	164.31	32.32	19.67	32	137.19	38.71	28.22
Ten months	13	189.23	29.19	15.43	33	159.27	37.71	23.68
Eleven months	12	197.25	28.38	14.39	19	166.79	48.12	28.85
Twelve months	12	218.92	38.95	17.79	11	181.55	36.5	20.1
Thirteen months	11	225.36	29.05	12.89	9	202.33	40.83	20.1
Fourteen months	10	229.5	34.1	14.86	7	223.57	32.25	14.43
Fifteen months	10	226.7	41.1	18.13	2	195	....	......
Sixteen months	10	242.8	41.1	16.92	......	....	....	......

The outstanding fact of genetic importance is that the variability of the F_2 generation is distinctly greater than that of F_1. That, to be sure, is what was to be expected from the genetic standpoint, and it is in agreement with the experience of practical breeders. It is definite proof of two facts: First, some of the factors which have the power to stimulate growth are not identical in the Duroc-Jersey and the Berkshire breeds; second, there is some degree of homozygosis for these growth-stimulating factors within the limits of each breed. To what extent being pure-bred from the standpoint of the breed registry society indicates homozygosis for these growth factors; whether the number of factors involved can be determined; whether the differences of growth indicate differences in the identity of the factors or merely differences in the combinations; and whether these factors are so linked in groups as to make recombination of the desirable ones impossible, or at least impracticable—these are all interesting questions which these data raise but are too meager to answer.

Other interesting questions which are not, however, primarily genetic, relate to the shape of the curve. The maximum variability seems to be reached at or shortly following weaning time and coincides with the period of the lowest mortality. Does this mean that the best time to select breeding stock for size and early maturity is when they are from 3 to 6 months old? Of course it was impracticable to keep the hogs, which were not desired for breeding purposes, until they were 2 or 3 years old, and therefore there is still left open the question as to whether the hogs would not have finally become more uniform in size; that is, as to whether the differences were in potential size or in earliness of maturity.

Plate 2, K, is a photograph of one of the F_2 litters most extreme in its range of variation in growthiness. These four individuals had received identical treatment, and none of them had ever been recorded as sick, yet when this photograph was taken, when they were being marketed at the age of 11 months, the weights were, from left to right, 221, 293, 90, and 170 pounds. Possibly more animals are born runts than is commonly believed.

MAMMARY PATTERN

This subject was treated by the senior author in a detailed study (21) based upon many more data than are available in this experiment. Therefore, the present data will merely be briefly summarized in so far as they bear upon the following points, which are discussed in detail in the original study.

SEAT OF THE GREATEST VARIATION

The frequency of the occurrence of the nipples and the variations in their positions are presented in Table IX. The method of counting the position of the mammae is the same as that used in the original paper.

TABLE IX.—*Number and position of nipples of all pigs*

	Number and frequency of pair—						
	1	1½	2	2½	3	3½	4
Number of nipples	536	3	466	39	495	6	498
Frequency mammae	98. 52	. 56	86. 3	7. 22	90. 92	1. 11	92. 22
Frequency variations	1. 48	....	13. 7	....	9. 07	....	7. 78

	Number and frequency of pair—						
	4½	5	5½	6	6½	7	7½
Number of nipples	16	423	46	268	11	124	0
Frequency mammae	3. 29	86. 32	9. 39	81. 21	8. 33	93. 93	....
Frequency variations	....	13. 68	....	18. 79	....	6. 06	....

	Number and frequency of pair—		
	8	8½	Inguinal.
Number of nipples	3	1	536
Frequency mammae	75	25	98. 53
Frequency variations	25	......	1. 47

The second wild boar himself possessed but four pairs of mammae and the number of mammae in his offspring was so uniformly less than in animals of the domesticated breeds that the wild boars and all their descendants, which are included in Table IX, are grouped separately in Table X.

TABLE X.—*Number and position of nipples of wild boars and descendants*

	Number and frequency of pair—						
	1	1½	2	2½	3	3½	4
Number of nipples	111	....	99	6	104	1	96
Frequency mammae	99. 1	....	88. 39	5. 36	92. 86	. 9	88. 89
Frequency variations	. 9	....	11. 61	....	7. 14	....	11. 11

TABLE X.—*Number and position of nipples of wild boars and descendants*—Continued.

	Number and frequency of pair—				
	4½	5	5½	6	Inguinal
Number of nipples	7	58	2	7	111
Frequency mammae	6.48	85.29	2.94	87.5	99.1
Frequency variations	....	14.71	....	12.5	.9

Evidently the smaller number of mammae possessed by the wild race is inherited to some extent, but whether as an independent character or because correlated with other body structures is not disclosed by these data.

Using the data in Table VIII and arranging the pairs in the order of frequency of variation, the following sequence results: Sixth, second, fifth, third, fourth, seventh, and inguinal. (The eighth was discarded because of small numbers.) This sequence duplicates exactly that found in the original study, except that there was no seventh pair in those animals.

The rank of the point of most frequent appearance of the triangle pattern is as follows:

	Percentage of total.	Percentage of pigs of the wild cross.
Between fifth and sixth	9.39	2.94
Between sixth and seventh	8.33	....
Between second and third	7.22	5.36
Between fourth and fifth	3.29	6.48
Between third and fourth	1.11	.9
Between first and second	.56	....

This agrees almost exactly with the previous results. (The triangle at 8½ is not included because of the small numbers.)

The rank of the point of most frequent suppression of a nipple was as follows:

	Percentage of total.	Percentage of pigs of the wild cross.
Second pair	5.33	6.25
Fourth pair	3.89	3.70
Sixth pair	1.82	....
Fifth pair	1.02	1.47
First pair	.74	.89
Third pair	.55	.89
Inguinal pair	.36	.89

While this does not agree exactly with the previous results, still no pair is further than two places from its original rank, and, in general, the order is similar.

RELATION BETWEEN NUMBER OF MAMMAE AND ASYMMETRICAL VARIATIONS

To determine the relation between the number of mammae and the frequency of the suppressed nipple or the triangle variations, the animals were grouped as shown in Table XI. Twelve animals possessed both variations and are counted twice.

TABLE XI.—*Relation between number of mammae and frequency of suppressed nipple*

	Pair No. 4.	Pair No. 5.	Pair No. 6.	Pair No. 7.	Pair No. 8.	Pair No. 9.
Number of animals of each class with all even pairs of mammae	2	26	42	51	5	0
Per cent	100	96.3	51.85	36.43	11.36	....
Number of animals of each class with triangle	....	....	21	68	20	2
Per cent	....	....	25.93	48.59	47.5	67
Number of animals of each class with suppressed mammae	....	1	18	21	19	1
Per cent	....	3.7	22.22	15.0	45.45	33

The increased percentage of animals with two types of variations among the animals with a larger number of mammae speaks for itself.

INHERITANCE OF THE TWO FORMS OF VARIATION

Nothing could be learned in regard to this point from these data because of the smallness of numbers and because the only boars with any considerable number of offspring—the second wild boar and the F_1 boar of the Berkshire×Duroc Jersey cross—lacked both variations. The only female having a very large number of offspring—the F_1 Duroc-Jersey sow—possessed the suppressed mammae herself, but produced some offspring having an even pattern, others having a triangle, others with a suppressed mammae, and others with both variations when mated to her son with an even pattern, and still others with a triangle when mated to a Berkshire boar whose pattern is not recorded.

RUDIMENTARY MAMMAE TO THE REAR OF THE INGUINAL PAIR

The possession of this pair of mammae which is low upon the scrotum of the male or upon the inner part of the rear thighs of the female, has been described (*21*) as a sex-limited character due to a factor which is dominant in males but recessive in females, and as a sex-linked character (*22*) for which the male is the simple X sex. However, it appears that the latter interpretation is faulty in view of certain data presented in the original article (*22*) in which this interpretation was advanced. These data are those which show that there resulted from the mating of boars with rudimentaries to sows with rudimentaries, offspring in the ratio of two boars possessing rudimentaries to one female possessing

them to one female lacking them. The occurrence of the female lacking rudimentaries appears inexplicable upon the sex-linked basis, and to that extent the original sex-limited interpretation appears more nearly correct. This is especially true because the only published evidence against it has been the purely negative evidence of failure to find a boar homozygous for the factor for rudimentary mammae. The sex-linked interpretation is therefore abandoned.

However, the data disclosed in the present experiments do not completely support the sex-limited interpretation, although clearly indicating that the character is in some way involved with sex. The authors are somewhat at a loss to know how to regard these data. The aberrant individuals indicated in Table XII were recorded by three separate persons, none of whom was the senior author, who had had the greatest experience in recognition of the character. The fact of his absence in the military service made it impossible for him to check the records, hence they are presented for what they are worth in the belief that their publication is less open to criticism than their suppression. With the exception of one female recorded as possessing rudimentaries which is discussed later, it will be noted that the aberrant animals possibly result from errors of omission rather than of commission, hence it is probable they represent a lack of uniformity in observation among the different people connected with recording the data of the study. The data as recorded from the different types of matings are given in Table XII.

TABLE XII.—*Distribution of rudimentaries in the offspring*

Character of parents.	Number of matings included.	Number of litters included.	Males.		Females.	
			With.	Without.	With.	Without.
Both with rudimentaries	9	16	56	**21**	33	37
Both without rudimentaries	4	5	....	12	....	22
Male with, female without	1	4	8	11	1	10
Male without, female with	1	2	1	**4**	1 (?)	11

The numbers in bold-faced type are those which were unexpected according to the original sex-limited interpretation of the inheritance of this character. The female with rudimentaries produced in the fourth type of mating is quite certainly wrongly classified, for the record shows her to have only one rudimentary and a mammary pattern so irregular that it is probable the one described as a rudimentary is really the last mamma of the abdominal series.

Omitting the questionable female from consideration, the following discussion is offered of the results in Table XII. Except for one litter in the first type of mating, all litters in the first and third types were sired by the same male, the F_1 Berkshire-Duroc-Jersey boar, and the dam of the one mating of the third type and the dams of seven of the nine matings of the first type were all full sisters to each other and to the boar. In spite of this close relationship, these matings when considered individually do not show a normal range of distribution in the ratios of males with rudimentaries to males without. Instead, four of the nine matings in the first type show approximate equality of males

with and males without rudimentaries. The other five matings show altogether only 2 males without the rudimentaries and 32 with the character. The data do not furnish critical evidence as to whether there are different genotypes among the females or whether there is merely a quite abnormal distribution. The numbers, of course, are not large, but they approximate so closely a simple 3 to 1 ratio for the males and a simple 1 to 1 ratio for the females from the first type of mating that they suggest strongly that the true explanation must be simple.

All the animals of the first and third types were entirely Berkshire and Duroc-Jersey in their ancestry. Three matings of the second type were entirely Tamworth and wild in their ancestry, and the fourth mating of this type was between a pure-bred Berkshire boar and a pure-bred Tamworth sow. The only mating of the fourth type was between a pure-bred Berkshire sow and the wild boar. Hence it appears at least possible that there may exist differences between the breeds in respect to the factor complex which cause this pair of mammae to be present. Thus, the matings of the second and fourth types, which produce the small percentages of progeny with rudimentary mammae (or none at all) include all the matings in which either Tamworth or wild blood is involved. Likewise all the matings in the first and third types involve only Duroc-Jersey and Berkshire blood, most of them being matings between F^1 individuals. It is worthy of note that the original theory of sex-limited inheritance of this character was based almost entirely upon data obtained from high-grade or pure-bred Duroc-Jersey animals (*21*).

It is not possible from these data to show clearly which of the expressions of the character is the dominant one. From the evidence of the matings of the first and second types it would seem that the possession of the rudimentaries was the dominant form of the character, since parents lacking them produce only offspring like themselves while parents possessing them produce both kinds of offspring. However, such an hypothesis entirely fails to explain the great preponderance of progeny without the character in the matings of the third and fourth types.

Since the numbers of animals in this study are so much smaller than in those previously reported by the senior author, and since there was so much confusion incidental to the entering into the military service of three of the four men connected with the records on this work, it is believed best not to consider the evidence critical until further data are secured which either support or discount the present records.

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PLATE 1

A.—The second wild boar used in these experiments. (Courtesy of the Iowa Agricultural Experiment Station.)

B.—Two pure-bred Tamworth sows and a litter out of one of them by a wild boar. The pigs are about 6 weeks old.

C.—The F_1 boar whose ears were not erect. Not used in breeding. Berkshire × Duroc-Jersey cross.

D.—A Berkshire × Duroc-Jersey F_1 barrow showing the almost complete dominance of the Berkshire shape of ear and face.

E.—The Duroc-Jersey sow that was the dam of all the F_1 breeding animals in the Berkshire × Duroc-Jersey cross.

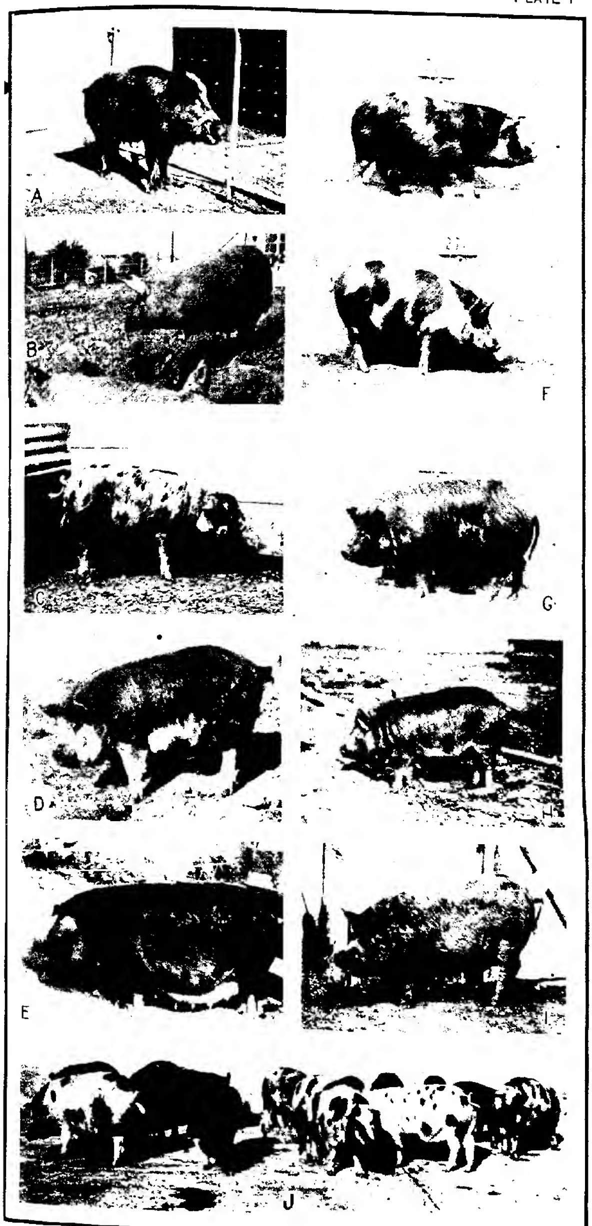
F.—Two F_2 individuals showing the segregation of ear shapes. Berkshire × Duroc-Jersey cross.

G.—The "pompadour" Berkshire type of forehead as seen on an F_2 individual of the Berkshire × Duroc-Jersey cross.

H.—An F_1 barrow with exceptionally straight face. Berkshire × Duroc-Jersey cross.

I.—A particularly long-faced individual. F_1 generation of Berkshire × Duroc-Jersey cross.

J.—Two F_2 litters of the Berkshire × Duroc-Jersey cross, showing the diversity of colors produced.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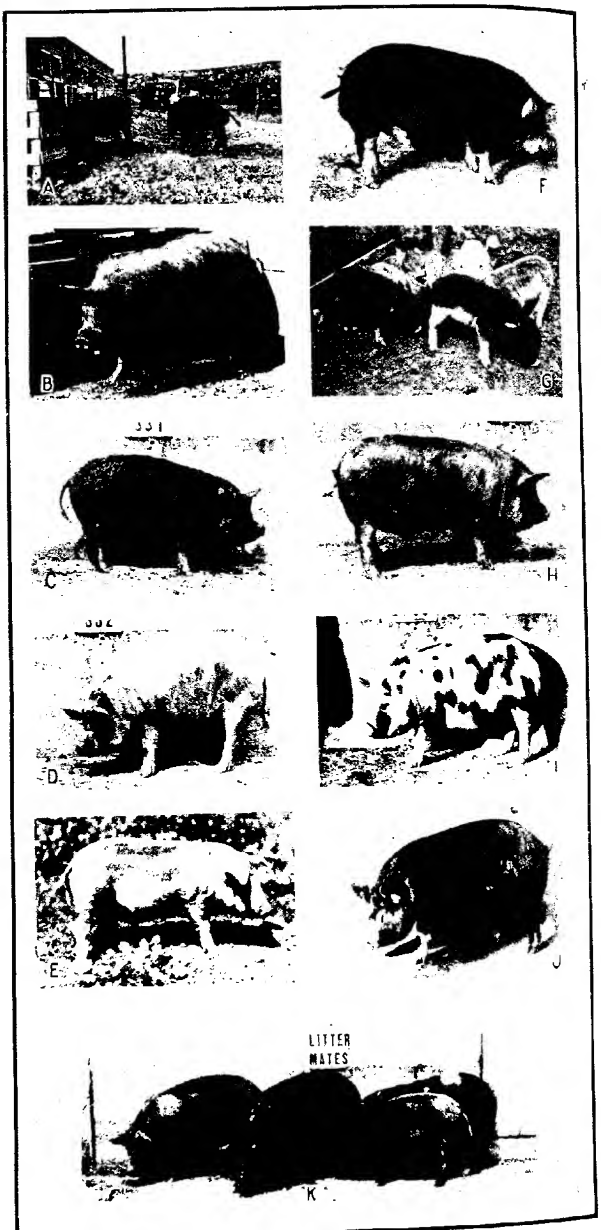
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PLATE 2

A.—The F_1 wild × Tamworth boar and the second wild boar.

B.—The F_1 boar, sire of all the F_2 pigs of the Berkshire × Duroc-Jersey cross.

C.—A self-red F_2 barrow. Berkshire × Duroc-Jersey cross.

D.—An F_2 barrow, red with a sandy belly when born, white when mature. Berkshire × Duroc-Jersey cross.

E.—A common type of "reversion." A striped pig of ordinary mixed domestic ancestry. (Courtesy of the Department of Genetics, University of Wisconsin).

F.—An F_2 boar, red with a sandy belly. Berkshire × Duroc-Jersey cross.

G.—An F_3 litter. Eight pigs of the 11 which showed stripes and light bellies. Berkshire × Duroc-Jersey cross.

H.—A red-and-black F_2 pig showing the average amount of black. Berkshire × Duroc-Jersey cross.

I.—A black-and-white F_2 pig showing the average amount of black. Berkshire × Duroc-Jersey cross.

J.—The black-and-white F_2 pig which most nearly approached the Berkshire standard as to the amount of black. Berkshire × Duroc-Jersey cross.

K.—Four litter mates from the F_2 generation. Note the variation in size. Berkhire × Duroc-Jersey cross.

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